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THE TECHNIC OF COLPOPERINEORRHAPHY

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THE surgical cure of rectocele requires an operation that affords an ample dissection of the lesion, its correction by plication or rectopexy, the suturing of the levators to the side of the rectum, the obliteration of dead space, the exact approximation of the vaginal mucous membrane, and an adequate repair of the perineum. These requirements have been repeatedly noted by numerous authors and their achievement is the objective of all operators.

Annoying hemorrhage, however, is frequent and at times prevents the surgeon from adequately carrying out his technic. It is the purpose of this

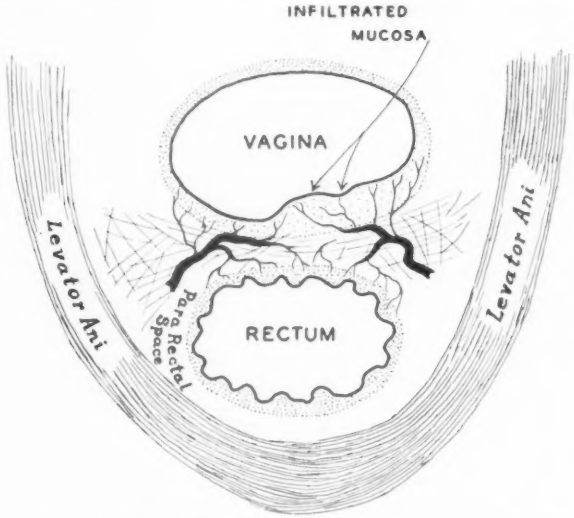


FIG. 1.—Diagram showing location of blood vessels in the wall of the vagina and the effect of infiltration of the field with adrenalin in novocain.

paper to present a method of dissection, based on anatomic considerations, which will do much to control the hemorrhage and thereby facilitate the operation.

Many of the blood vessels supplying the posterior wall of the vagina run in a rather definite bundle in the fascia. Their course is obliquely downward and inward, and they can be seen in the vaginal fascia, on the posterolateral aspect, about three-fourths to one and one-fourth inches from the introitus.

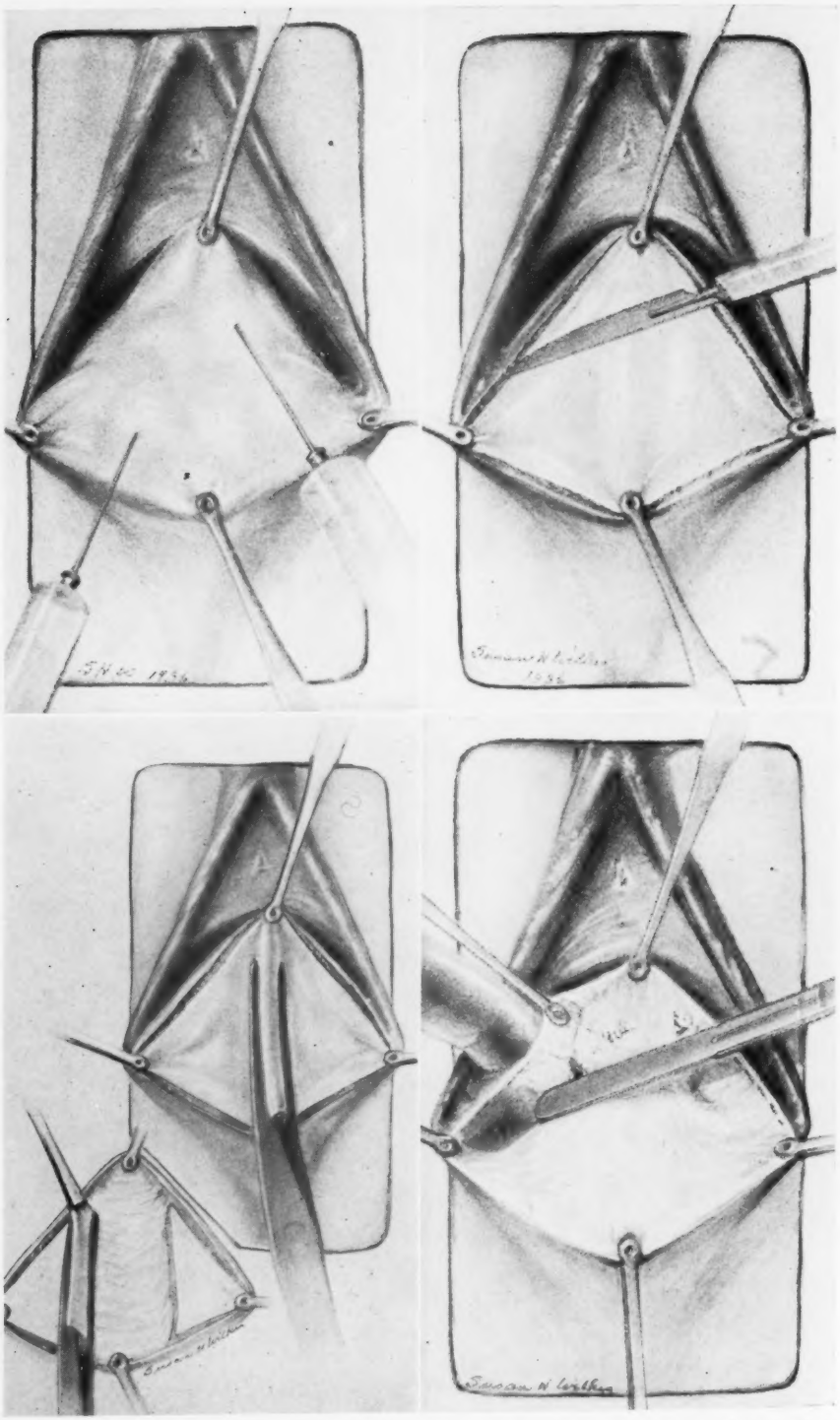
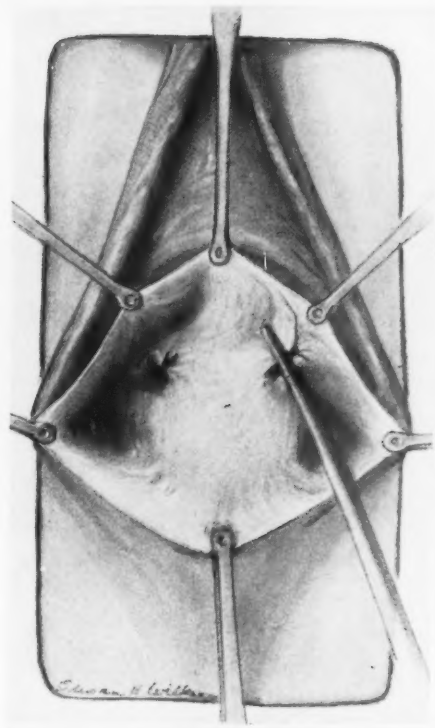


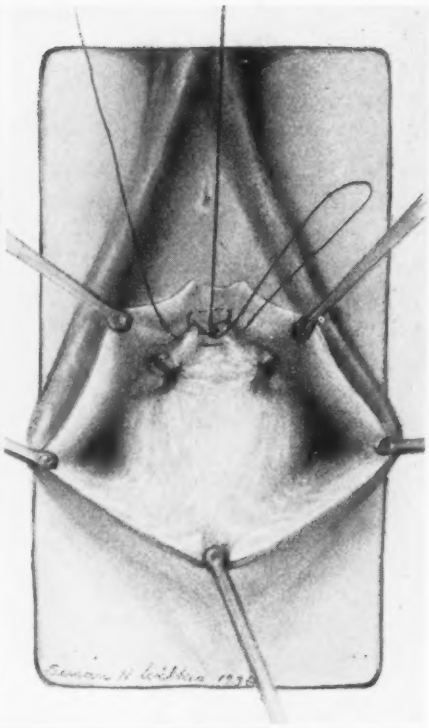
FIG. 2.—The rectocele is caught with clips and the field infiltrated with adrenalin in novocain.

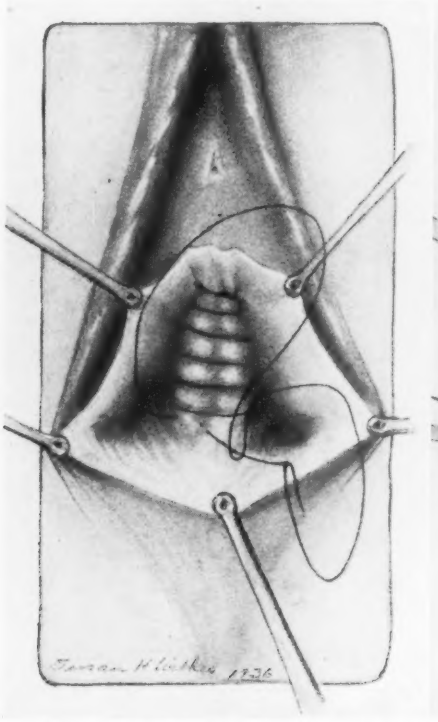
FIG. 3.—The area to be denuded is outlined with the scalpel.

FIG. 4.—The mucous membrane is removed with scissors.

FIG. 5.—The vessels are identified. The pararectal space is opened and the levators are exposed.







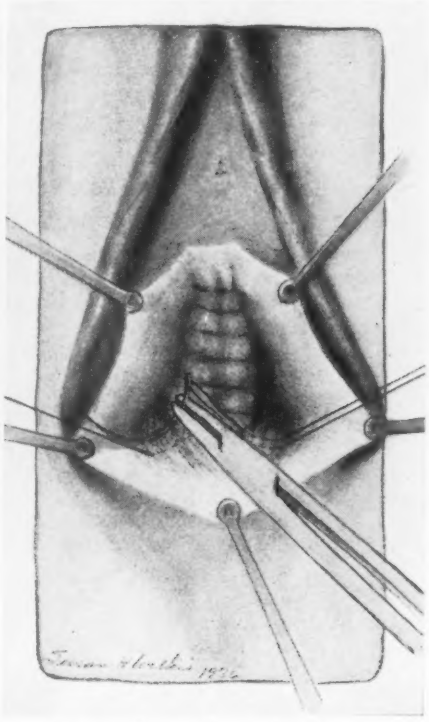


FIG. 6.—The vessels on the right side are ligated and the rectum is pushed back from edge of the mucous membrane. A clamp is shown on the vessels on the left side.

FIG. 8.—Stitch is continued, taking wide bites at the side of the rectum and plicating the rectum through the exposed portion. This obliterates the rectocele.

FIG. 7.—Suture is passed, taking a small bite of the rectum, going through the vaginal mucous membrane, crossing the midline, penetrating the vaginal mucous membrane, and taking a bite of the rectum. It is tied in the midline.

FIG. 9.—The levator sutures are passed.

They run toward the midline, breaking up into many small branches before they penetrate the fascia on their way to the mucosa (Fig. 1). It is obvious that, if one can remove the mucous membrane without going deeply into the fascia, the larger vessels will not be opened. A thorough preliminary injection of the field with adrenalin in novocain will separate the mucous membrane from the fascia and check the blood flow from the smaller vessels. The mucous membrane can be removed with scissors, after which the field is so dry that the vessels can be identified and ligated before exposing the sides of the rectum. The ligation of these vessels greatly decreases the

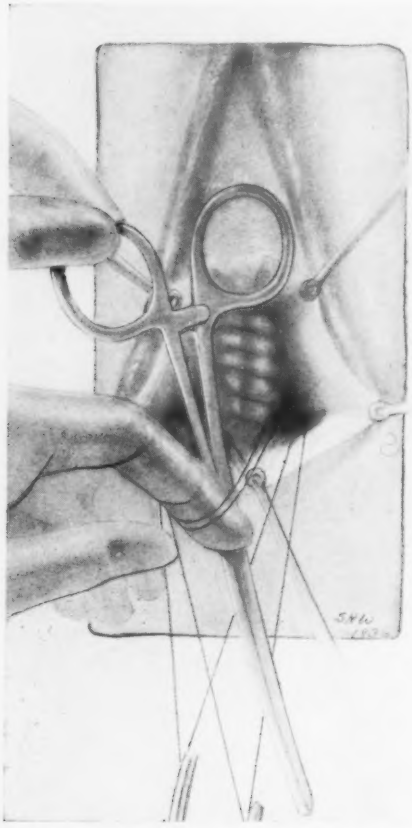


FIG. 10.—The inner portion of levator sutures are hooked upon the finger, withdrawn from the wound, and held by engaging the handle of a clamp in them.

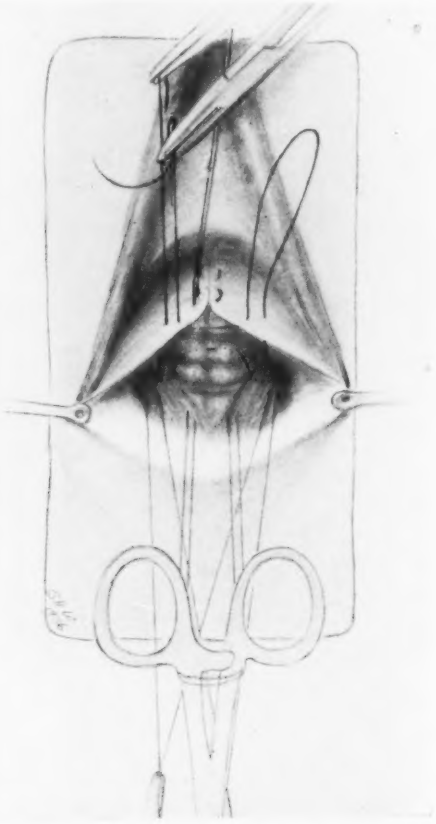


FIG. 11.—The first two on-edge mattress sutures are tied. The deep portion of the third suture penetrates the vaginal mucous membrane, takes a bite of the levator fascia, is reefed through the top of the rectum and the levator fascia of the opposite side, and penetrates the vaginal mucosa.

hemorrhage and allows the operation to proceed smoothly. The details of the dissection will be found in the following description.

Operative Technic.—(1) The mucous membrane of the vagina is caught with mucosa clips at the carunculae myrtiformes, at the top of the rectocele, and in the midline at the mucocutaneous junction. The field, as well as the area adjacent to it, is thoroughly infiltrated with adrenalin (1:120,000) in ¼ per cent novocain (Fig. 2).

(2) The area to be denuded is outlined with a scalpel (Fig. 3).

(3) The mucous membrane is removed with scissors (Fig. 4).

(4) The vessels are identified. A mucosa clip, placed on the mucous membrane above the vessels, draws it toward the symphysis. A finger is placed on the mucous membrane behind the clip and pushes its under surface into view. The vaginal fascia at this point is incised with a scalpel and, after very gentle dissection with the knife handle, a finger will open the pararectal space. The levators are well exposed laterally and the vessels stand out superiorly and medially. These are clamped and cut (Fig. 5).

(5) The vaginal fascia is now incised superiorly to the vessels and the rectum is pushed back about one-half inch from the edge of the mucous membrane. The entire field is now exposed, and the rectocele can be reduced by plication or rectopexy (Fig. 6). Plication will be described.

(6) A needle, armed with No. 1 chromic catgut, enters the fascia at the left side of the rectum, penetrates the vaginal mucous membrane in an upward direction, crosses the midline, penetrates the mucous membrane in a downward direction, grasps the fascia at the right side of the rectum, and is tied (Fig. 7).

(7) The suture is continued, plicating the rectum, and is tied at the lower angle of the wound. This obliterates the rectocele (Fig. 8).

(8) The levator sutures are passed, and the ends clamped (Fig. 9).

(9) The inner portion of the levator sutures are hooked upon the finger, withdrawn from the wound, and held by engaging the handle of a clamp in them (Fig. 10).

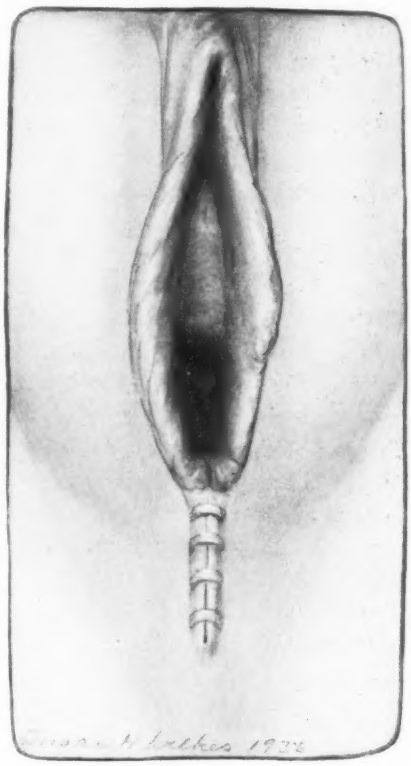


FIG. 12.—The trigone is closed with interrupted sutures and the skin is closed with clips.

(10) The sides of the rectum and levator are reapposed by the use of interrupted on-edge mattress sutures of No. 1 chromic catgut. This suture obliterates the dead space and adequately approximates the vaginal mucous membrane without inversion. In passing the suture, a needle is threaded at both ends of the suture. One needle enters the mucous membrane three-eighths of an inch from the edge, picks up the fascia at the left side of the rectum, passes over the rectum (picking it up at one or more points), grasps the fascia at the opposite side, and then passes out through the mucous membrane. The other needle is passed through the edge of the mucous membrane, on each side, and the suture is tied. Sutures are inserted in this manner until the incision in the mucous membrane is closed (Fig. 11).

(11) The levator sutures are tied, the trigone is closed with interrupted sutures of plain No. 1 catgut, and the skin is closed with clips (Fig. 12).

ECHINOCOCCUS DISEASE

REPORT OF A CASE OF PRIMARY ECHINOCOCCUS CYST OF THE UTERUS

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ECHINOCOCCUS or hydatid disease, recognized since the time of Hippocrates, is caused by the Taenia echinococcus, a minute tapeworm, two and one-half to five millimeters long, which finds its host in the intestines of the dog, and more rarely, of the cat, the jackal, and the wolf. The larvae are excreted in the feces of these animals, and vegetation and unprotected water supplies thereby contaminated. The wind-blown dust and the running streams carry the eggs, microscopic in size, far and near. Human beings, domestic animals, grazing cattle, and swine become the intermediate hosts in the parasitic cycle by ingestion of the egg embryos, or by direct contact with infected animals. Davis and Balboni[1] state that 27 species of mammals have been reported as harboring echinococcus disease, also that the dog is not itself subject to the hydatid form which is the larval stage and occurs only in the intermediate host. If the dog is fed upon flesh or viscera containing hydatid cysts, it develops in turn an adult tapeworm and the cycle is completed. However, as Magath[2] and others report, proof of the presence of the adult type in the dog is rare in North America, and the only authentic record is that of Curtice who found it in one dog at Washington, D. C. Von Siebold was the first to rear Taenia echinococcus experimentally in the dog by feeding it hydatids removed from cattle.

When the eggs of the Taenia echinococcus are swallowed by the intermediate host, the embryos are set free by the digestive processes, burrow by means of hooklets into the walls of the stomach or intestines, and are carried by the systemic circulation, or as some authorities believe, by lymph channels, to various parts of the body. When the embryo reaches its final destination in the liver, heart, lungs, *etc.*, the hooklets disappear and the embryo is gradually transformed into a cyst with two layers, containing a clear fluid, and wholly enveloped by a fibrous covering. From the internal germinative layer, daughter cysts develop inside of which are brood capsules each composed of two or more heads or scolices capable of becoming a tapeworm if excreted by the secondary host and ingested by the parent host, the dog. And from the daughter cysts, granddaughter cysts form, until the whole mass may become as large as a child's head.

There are two types of cysts: Echinococcus unilocularis; and echinococcus multilocularis, the latter being the rarer type.

The disease is a menace to public health in many sections of the world: Pomerania, Mecklenburg, Bavaria, Switzerland, certain provinces of Italy,

parts of France, Greece, Armenia, Yugoslavia, the steppes of Russia, and Turkey. In Iceland, it is the cause of one-seventh of the deaths. In Australia and New Zealand it is quite common. Barnett,[3] in a statistical survey of hydatid disease in New Zealand for the period of 43 years ending in 1934, estimated 3,550 cases, with the deaths for the whole Dominion, 534, and a mortality rate of 15 per cent. In a personal communication to the authors, he states that in New Zealand with a population of 1,500,000 there are approximately 125 new cases of hydatid infestation every year. A Hydatid Registry has been established under the auspices of the Royal Australasian College of Surgeons to record and tabulate the cases, and to date approximately 1,000 cases have been so registered and analyzed and about 200 additional cases have been briefly noted.

In the Argentine Republic, Uruguay, and southern Brazil, the incidence is high and all leading surgeons in Buenos Aires and Montevideo are thoroughly versed on echinococcus cysts. In 1922, the Argentine Medical Association devoted an entire week to the study and discussion of hydatid disease and published an extensive bibliography. In 1932, Felix Devé, Professor of Medicine at the University of Rouen, one of the foremost authorities on the disease, delivered a series of lectures on echinococcosis by special invitation of the Medical Association of Buenos Aires.

In North America, echinococcus disease is more prevalent than commonly supposed, and although the literature from Canada and the United States is not so voluminous as that from other parts of the world, a great number of cases have been reported. Lyon,[4] in 1902, assembled 241 cases. In 1921, Magath (in addition to 68 previously reported cases) cited 25 cases from The Mayo Clinic. And in 1927, Mills[5] added 63 hitherto unpublished records. In all, the literature since 1902, through the year 1935, contains reports of 268 cases, making the grand total of reported incidence 509 cases. And the fact that there were 155 deaths from hydatid cysts (111 of the liver, 44 of other organs) in the United States during the five years ending in 1934 makes it extremely probable that there were as well a goodly number of cases not reported in scientific articles.

In the past, it has been persistently argued that the incidence in North America was only among foreigners or those who had lived in countries where the infestation was rife. The case we are reporting was in a Negress, who not only was native born, but had never been outside the state, living always near Alexandria, La., at first 20 miles north of that city and more recently the same distance south of it; nor had she ever been in intimate contact with a person from outside the state. In Lyon's review of 241 cases, 135 were among foreigners, 92 nationality not stated, and of the remaining 13, ten were Negroes, two Canadians, and one American. Of the 268 cases since 1902, there were 41 whose nativity was not known, 26 Americans, and two Canadians, or 12.3 per cent native born, and 199 foreigners, or 87.7 per cent (Table I).

TABLE I

NATIVITY OF THE 268 CASES REPORTED SINCE 1902

Albania	1	Poland	1
Armenia	8	Portugal	2
Austria	10	Roumania	1
Cape Verde Islands	1	Russia	13
England	12	Spain	2
Finland	1	South America	3
France	2	Syria	2
Germany	11	Sweden	1
Greece	36	Turkey	2
Hungary	1	Wales	3
Iceland	15	Yugoslavia	2
Ireland	3	Foreigner (country unknown)	4
Italy	58	Canada	2
Mexico	1	United States	26
New Zealand	2	Not known	41

Native born ..... 12.3 per cent
Foreign born ..... 87.7 per cent

Riley[6] believes that the assertion of a number of workers that the majority of cases occur in foriegn born individuals, or those who have traveled in endemic regions, is not a sufficient explanation in view of the frequency of the disease in domestic animals. And this seems plausible, for echinococcus infestation occurs in hogs, sheep, and cattle in alarming numbers in Virginia, Oklahoma, and Arkansas.

It has been impossible to get any statistics from the Federal Government or the packing houses as to the incidence of the disease in animals slaughtered throughout the country, other than that (from the Bureau of Animal Industry, Washington, D. C.) during the fiscal year ended June 30, 1936, there were reported condemnations of 1,513 cattle livers and 21 calf livers on account of echinococcus infestation. Therefore, it is easily possible that in districts where the meat supplies are not carefully guarded, and hogs, sheep, and cattle are home killed and the offal is thrown to dogs to eat, the vicious circle of infestation is fostered and encouraged and the human incidence increased.

When echinococcus disease travels by its route of contamination from animal to man, the liver for anatomic reasons is the site most often invaded.

TABLE II

PERCENTAGE OF INCIDENCE OF ECHINOCOCCUS AT VARIOUS SITES IN MAN

Author	Liver	Lungs	Other Organs	Abdominal Cavity	Skin and Muscles	Brain	Kidney	Spleen	Bone	Heart
Devé	74.9	8.5	4.2		5.7	1.4	2.1	2.3	0.9	
Thomas[7]	57.0	11.6	18.4			4.4	4.7	2.1		1.8
Lyon	73.7									
Beckman[8]	75.0	9.0	16.0							
Stitt[9]	60.0	10.0	8.0	7.0	7.0	3.0	3.0	2.0		
Mills[10]	75.0									
Barnett	70.0	10.0	20.0							
Magath	76.3									

Various writers have estimated the percentages of incidence, as shown in Table II.

Analysis of the 268 cases recorded since 1902 shows the location of the cysts to be as noted in Table III:

TABLE III

LOCATION OF CYSTS IN THE 268 CASES RECORDED SINCE 1902

Organ	Cases	Percentage
Liver	212	69.7
Lungs	22	7.0
Kidney	17	5.5
Bladder	1	0.3
Abdomen	8	2.6
Peritoneal cavity	21	6.8
Pelvis	4	1.3
Spleen	4	1.3
Retroperitoneal	2	0.6
Heart	2	0.6
Pleural cavity	4	1.3
Gallbladder (2) and bile duct (1)	3	0.9
Neck	1	0.3
Bones	3	0.9
Brain	1	0.3
Prostate	1	0.3
Spinal cord	1	0.3

Echinococcus cysts of the female pelvis and uterus have been noted from time to time in the foreign literature. However, some of these upon investigation appear to have been not true echinococcus cysts but hydatiform moles. Confusion between the two seems frequent in the literature of the last decades of the nineteenth century and in the cases cited during that period, in the absence of microscopic confirmation, there is always doubt as to validity. In 1891, a case of echinococcus cyst of the uterus was reported by Altormyan[11] from Aleppo, Syria. John Davies Thomas quotes Neisser's report of 35 hydatids found in the true pelvis, 20 in females, 15 in males; besides these there were 27 cases in which the uterus or ovary was considered to have been the seat of the parasite. Thomas also mentions the report of Schatz giving 66 instances of hydatid disease in the female pelvis, as well as a case recorded by Allen of a large hydatid cyst found at autopsy "in the pouch between the bladder and the uterus"; and another case "attached to the peritoneum covering the fundus of the uterus and the bladder. In Thomas' own series the uterus is credited with 29 instances and the ovary with 16. He also gives a list of 72 pelvic cases, 14 in the uterine wall, eight in the ovary, and seven in the broad ligament. Unfortunately, no definite references are given upon which to base the authenticity of Thomas' statistics; his book is a collection of lectures and there are gaps in the context, as some of the data were lost previous to compilation of the book.

During the present century also, echinococcus cysts of the female genera-

tive organs have been reported from abroad. Mills cites one from the Argentines (Cuneo) of an echinococcus cyst of the ovary in a girl, age 15; and another (Chueco) of the posterior wall of the uterus in a virgin, age 15. Cuff[12] reports an echinococcus cyst of the uterus from Cyprus. The literatures of France, Germany, and Scandinavia also contain references to echinococcus cysts of the uterus. The conclusion is, therefore, that it is not an unknown condition, but is by no means common, and the rate of incidence cannot be compared with that in other organs. As a rule, the cyst of the uterus is secondary to affectation of the liver, kidneys, *etc.* Turenne[13] states that primary hydatid cysts of the uterus are so rare that he has only been able to find 10 in the literature, in addition to the one he personally reports; and that Mercade found 14 cases, of which only eight were primary cysts of the uterus. Barnett, in a recent personal communication, agrees that primary hydatid cysts of the uterus are extremely rare, probably as rare as those occurring in the thyroid gland, the heart, or the prostate. In the Hydatid Registry cases there is only one that can without any doubt be classed as primarily uterine:

Case 952.—Found by Dr. K. Christie, July 5, 1936, at operation for uterine fibroids. Two large fibroids were removed; a third mass the size of an orange embedded in the anterior wall of the uterus, between the uterus and the bladder, was also excised on the assumption that it was another fibroid, but subsequent examination showed it to be a hydatid cyst containing the typical fluid and daughter cysts.

In North America, very few echinococcus cysts of the female pelvis are recorded. Lyon includes three in his review, and McKechnie[14] and Kerwin[15] each report a case (Table IV).

TABLE IV

ECHINOCOCCUS CYSTS OF THE FEMALE PELVIS REPORTED FROM NORTH AMERICA

Case	Location	Observer
Female, age 73, from Albany, N. Y.	Liver and ovary	Low (1822)
Female, from Toronto, Ont.	In walls of pelvis, two cysts in liver, rupturing into intestine	Cameron (1882)
German female from Argyle, Ill.	Bladder, pelvis, liver, spleen, kidney, omentum, peritoneum, diaphragm, and pericardium	Allaben (1891)
Female, age 40, born in Iceland	Pelvis and bladder	McKechnie (1910)
Female, age 44, born in U. S.	Pelvic peritoneum	Kerwin (1916)

Of the cases of echinococcus cyst of the uterus reported from North America, none is proven primary, and some are obviously spurious. Lyon omits all but one (Van Giesen) from his review as of doubtful validity, and the histories indicate very positively that the cysts were of chorionic and not of parasitic growth (Table V).

We, therefore, believe there is justification for our claim that we are reporting the first case of echinococcus cyst of the uterus in the United

TABLE V

ECHINOCOCCUS CYSTS OF THE UTERUS REPORTED FROM NORTH AMERICA

Platt (1847) "Hydatid in utero"	Included by Sommer but omitted by Lyon as of doubtful validity
MacNevn (1849) Cyst of uterus coexistent with pregnancy and expelled intact during labor	Not definitely proven parasitic. Not inincluded by Lyon
Smith (1856) "Hydatids in utero"	Included by Sommer but omitted by Lyon as of doubtful validity
Ford (1868) "Hydatids in utero"	Included by Sommer but omitted by Lyon as of doubtful validity
Henry (1871) No record except entry in report of Montreal General Hospital: "Hydatides uteri; discharged"	Of doubtful validity; not included by Lyon
Gross (1882) Hydatid of uterus	Not definitely proven parasitic. Not included by Lyon
Van Giesen (1887) "Multiple cysts: liver, omentum, abdominal wall, peritoneum, fascia of psoas muscle, mesentery, and uterus	Included by Lyon, but obviously not a primary cyst of the uterus

States, in a native born female, as well as the first primary echinococcus cyst of the uterus from North America.

Mills, Devé, and other authorities point out that the seeds of echinococcosis are sown in infancy and the majority of hydatid cysts cause no symptoms until the patient has attained the age of 20 to 40 years. Stitt notes that the growth of the cysts is slow and the course of the disease is protracted two to eight years. Davis and Balboni cite the case of Magnuson, who had had it under observation for 40 years. And, as Dew[16] puts it, the majority of cysts, as seen clinically, are nearly as old as their hosts. Whether infestation occurred in childhood in the present case is, of course, a matter for conjecture.

The symptoms of echinococcus cysts are those of any slowly growing tumor, and in the absence of complications of the liver, as in this case, there is a symptomless enlargement until the size of the cyst produces discomfort.

When echinococcus cysts are suspected before operation, it is sometimes possible to confirm the diagnosis by certain laboratory tests: the precipitin test of Fleig-Lisbonne; the complement fixation test of Bordet Gengou, which is based upon the Wassermann reaction and was first applied by Ghedini in 1906; and the Casoni skin reaction. But none of these tests is infallible, nor is roentgenologic diagnosis, nor is it always possible to elicit hydatid thrill or fremitus, said to be present in 30 per cent of the cases. A high eosinophilia is sometimes indicative; in the present case, eosinophiles were only 1 per cent.

The treatment of echinococcus cysts is purely surgical. Davis and Balboni believe the use of salvarsan, as proposed by Kolbe, is not effective and that tapping the cysts through the abdominal wall is to be condemned because of danger of leakage of the fluid and daughter cysts into the peri-

toneal cavity with dissemination of the disease. When possible, excision en masse is the procedure of choice, but this is very seldom feasible, especially in echinococcus cysts of the liver. Therefore, marsupialization, with or without postoperative drainage, must be resorted to. Briefly, the accepted method is to aspirate the cyst contents and then inject a 1 to 2 per cent formalin solution, leaving it in about five minutes, in order to kill the scolices. Dew recommends injecting enough pure formalin to make, with the fluid remaining in the cyst, at least a 1.5 per cent solution. Any redundant cyst wall is then excised and closure made. The "closed method" is usually employed in simple, nonsuppurating cysts. For complicated, suppurating cysts, marsupialization with drainage is the accepted procedure. In both procedures an additional precaution is often taken by attaching the suture line to the abdominal wall; this is the "operation of Posadas."

Case Report.—I. B. W., a colored female, age 22 years, born in the United States, was admitted to Charity Hospital, New Orleans, November 29, 1935, complaining of an abdominal mass. She had been conscious of the growing tumor for four years, following delivery, in 1931, with dystocia or postpartum problems, at the termination of an uneventful pregnancy. The mass increased rapidly in size for a time, and according to the patient, was larger at one time than another. During the months preceding operation growth was very slow. It had, however, become sufficiently large to cause a feeling of pressure and to give discomfort, particularly when the patient stooped over to pick cotton. The patient found that pressure could be relieved by lifting the mass out of the pelvis with her hands. Her menstruation, which began at 12, was four to five days in duration with slight dysmenorrhea always, and had for the past two or three years been accompanied by some flooding; there were no other menstrual disturbances.

Except for the tumor mass, physical examination was negative. Liver, spleen, and kidneys were not palpable. The tumor mass in the lower abdomen was about the size of a six months' pregnancy, ovoid in shape, smooth in outline, freely movable, and of a semifluctuant consistency. Some pain was experienced upon deep pressure in the left flank. The vaginal examination showed the outlet to be parous; Bartholin's and Skene's glands negative. No cystocele or rectocele. There was a unilateral, well healed laceration of the left side of the cervix. The tumor mass completely filled the pelvis and was apparently intimately attached to the uterus, as no definite uterine outline could be distinguished except that portion comprising the lower uterine segment. A diagnosis of myoma of the uterus was made.

Operation.—The abdomen was opened under ether anesthesia through a subumbilical midline incision about four inches in length. A cystic mass about the size of a large grapefruit presented itself and was easily delivered from the abdomen. This mass was attached to about one-third of the left anterior uterine wall by a sessile base approximately two inches in diameter, and the omentum was adherent to the cyst wall in one place. This adhesion was released. Both ovaries and the right tube were perfectly normal. The left tube had become attenuated and was stretched over a portion of the cyst wall to such an extent that it was difficult to identify this organ. It was quite evident that it would be impossible to remove the cyst without rupture unless a portion of the fundus of the uterus was removed. A partial fundectomy was therefore performed and the tumor in its entirety delivered without a spill. The appendix was macroscopically diseased and was removed.

Knowing that the pelvic cavity was an unusual location for such a cyst, general exploration was made in an attempt to discover a primary focus in the liver, kidneys, or spleen. The liver presented a sharp edge, was resilient to touch, with no nodula-

tions. There was a small lymph node palpable along the common duct near the foramen of Winslow. Both kidneys and the spleen were similarly explored with negative findings. The omentum was entirely negative except for the small adhesion to the cyst wall. After operation, roentgenologic examination was made to eliminate the likelihood of the presence of echinococcus cysts in the chest and bones, with negative findings.

Pathologic Examination.—Gross: The specimen revealed a large cystic mass attached to about one-third of the fundus of the uterus (Fig. 1). The walls of the cyst were thickened and showed irregular sized patches of lipoid degeneration. To one of these patches, a strand of omentum was strongly adherent. The left tube in its proximal half was plastered to the posterior wall of the cyst by edematous inflammatory adhesions. The tube was so spread out that it was almost impossible to identify it.

FIG. 1.—Gross specimen of the tumor removed. (A) Fundus of uterus. (B) Laminated membrane (C) Germinating layer.

Dissection of the tube showed the lumen intact with no evidence of any connection with the cyst cavity. An attempt was made to probe the uterine cavity to determine whether this connected with the cyst cavity. No communication could be established. The interstitial portion of the tube was patent, but only filiform in diameter. Grossly, the cyst seemed to be arising directly from the wall of the uterus and not formed by an adhesive process. The left round ligament led directly into the lateral wall of the cyst. The cyst wall was about one-third to one-half centimeter in thickness, fibrous, and edematous, with yellowish lipoid patches as described previously. The broad ligament was posterior and lateral to the cyst wall. On incising the cyst wall, a turbid, lemon-yellow colored fluid escaped, containing numerous thin walled cysts of all sizes, ranging from that of a small bead to a large marble. Many of the cysts showed thin walls and had a translucent appearance. Quite a number had a hazy, grayish, thick wall. The cyst cavity was lined by a thin granular, fragile, gelatinous membrane, on the inner surface of which numerous small cysts were attached. Many of these attached cysts were pin point in size. Smear from the cyst revealed hooklets and small cysts with activity (Figs. 2 and 3).

After the operative procedure, a more detailed history was sought to determine

the definite social status of the patient and the possible source and time of her infestation. As stated, she was born in Louisiana about 20 miles north of Alexandria and had never been outside of the state. Recently, she moved about the same distance south of Alexandria, but had always lived in these two localities and had never contacted intimately an individual from outside the state. She advised that she chewed tobacco regularly and had an occasional drink of spirits. She had always lived on a farm where sheep, goats, and cows were raised and constantly had dogs as pets and lived with them intimately. When she became aware that she was pregnant, she began to eat clay, and reported that this is the custom among women in that vicinity, in the belief that certain minerals are supplied by eating clay which have a good influence on the growth of the fetus and the health of the pregnancy. Approximately a cup of clay a day was ingested, taking care to select the clay from localities where a dog had not defecated. She preferred to eat the clay in a dry state and gathered most of her supply during the dry season. However, if she had to get the clay when it was wet,

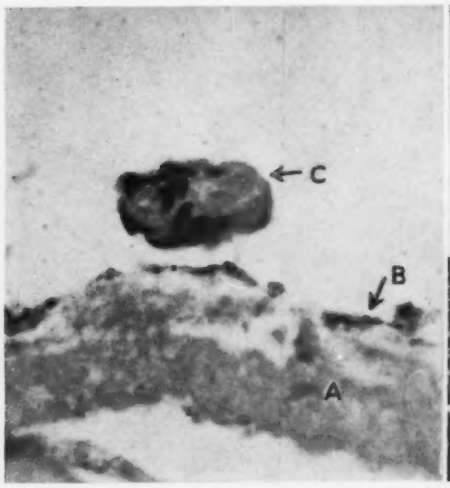

FIG. 2.—Section through the fundus and attached hydatid cyst wall. Myometrium (A) below and cyst wall (B) above. The greater part of the cyst wall is made up of the laminated membrane (B) which stains lightly, while the germinative layer (C) is distinguished as a thin dark layer lining the wall. (Low power.)

FIG. 3.—Portion of hydatid cyst wall. Below is the lightly staining laminated membrane (A) with the thin, darkly stained germinative (B) layer above. Free from the wall in this section is a degenerated scolex (C). (High power.)

she baked it in cakes in the stove before eating it. She also reported that she formed the habit of eating paper regularly; this also included pasteboard boxes. These perverted tastes became a fixed habit with the patient and began with the inception of her first and only pregnancy, in 1931.

The patient had an uneventful convalescence and was discharged December 19, 1935. Follow up nine months postoperatively revealed that she has remained in excellent health, her only complaint being a vaginal discharge. There has been no recurrence of the cyst and no discomfort in spite of hard work at cotton picking. She has, however, discontinued the diet of clay dust and paper.

SUMMARY

(1) Echinococcus disease is a public health problem in many parts of the world and is more prevalent in the United States than is realized.

(2) To date, 509 cases have been reported in the literature of the United States and Canada.

(3) Pelvic echinococcus disease is rarer than that of other organs and tissues.

(4) Of the cases of echinococcus cyst of the uterus reported in the literature of the United States and Canada, some are apparently erroneously classified and are in reality hydatiform moles. None of these cases is proven primary echinococcus cysts of the uterus.

(5) A case of primary echinococcus cyst of the uterus is herein reported, in a native born Negress, treated by partial fundectomy, with complete recovery, and no recurrence of the cyst.

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THE SHROPSHIRE SUPRAVAGINAL PLASTIC OPERATION

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DR. L. S. SHROPSHIRE,[1] in 1914, suggested a supravaginal plastic procedure in connection with an hysterectomy, as a result of which the morbidity was greatly reduced. The method impressed me so favorably that I have operated upon a number of cases following, in the main, his technic. The results have been so uniformly favorable that I have, from time to time, adopted the principles, as set forth by him, in procedures other than that of hysterectomy, and have been convinced of its merits. It seems to me that surgeons should give it more consideration than it has received thus far. Dr. H. H. Ogilvie,[2] of San Antonio, apparently is the only one who has, as yet, reported upon the employment of this technic, and cites 119 cases with only one death. Doctor Shropshire, in his original article, reported 15 cases. His communication is short enough to justify quoting it:

"I term this 'new' because as far as I know or have been able to ascertain, the method outlined has not been employed by anyone before, and it is

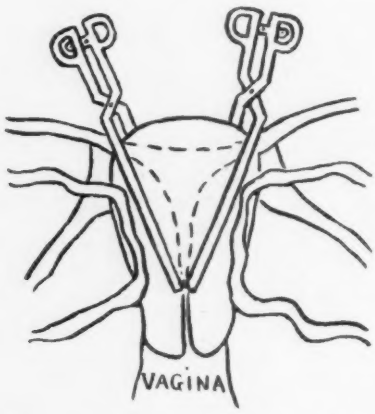

FIG. 1.—Showing the clamps in place.

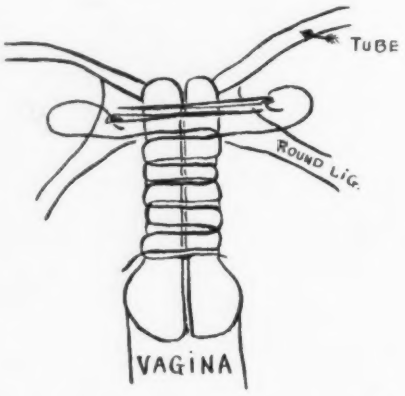

FIG. 2.—Showing the saddler's stitch as applied.

distinct from the flap method, which is commonly done. For a long time I have thought that the recovery after a hysterectomy was unnecessarily prolonged, and I have arrived at the conclusion that the impingement of the nerve trunks supplying the uterus and its appendages, within the ligatures used in tying off the adnexa before their separation from the organ is largely responsible for it. To avoid this slow recovery and to prevent the many reflex disturbances which are manifest for so long a time after the operation I have devised the following method of procedure: After separating the bladder from the uterus down to the internal os, I clamp the uterus on either side from the insertion of the fallopian tube to its center at the internal os with a specially devised hysterectomy clamp, and using my

clamp as a guide, I transfix the uterus at the points of the clamps with a long sharp-pointed knife, bringing the blade out at the fundus at the inner side of the clamp, making a smooth-cut surface; then by drawing my clamps together I bring the two marginal-cut surfaces of the uterus into close apposition. With a long No. 2 chromic gut suture, with needle on either end, I begin at the points of the clamps by passing one needle between the blades of the clamp on one side and drawing the suture through to its center, then by inserting a needle from either side make a saddler's stitch until I reach the top of my flaps, when the sutures are tied securely. After this the clamps are removed and with a No. 2 chromic gut I begin at the lower end of the cut surfaces, make a deep running suture up the anterior surface, over and down the posterior surface opposite the starting point. With a fine catgut suture I stitch the bladder to this stump to hold it up in its normal position.

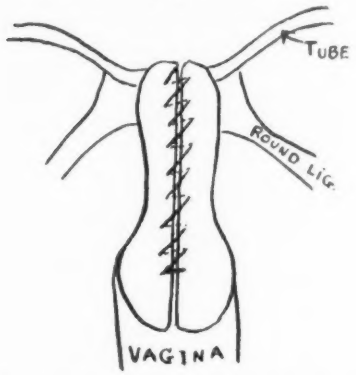

FIG. 3.—Showing appearance of the stump after the running suture is introduced.

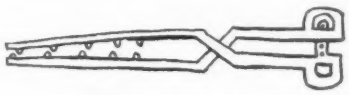

FIG. 4.—Showing a specially devised clamp, with five teeth on either jaw of the clamp to prevent it from slipping off the uterus. The jaws of the clamp must be at least one inch apart at the joint.

"I deem it worth while to mention only a few of the very apparent advantages of this operation over the old method. I claim for this operation that it avoids the tying off of any nerve trunks or the destruction of any important tissues—it takes away most of the body of the uterus and all its endometrium. It is indicated in any hysterectomy except for malignant degeneration. In cases of fibroid tumor, where my especially devised clamp cannot be used, I clamp the adnexa with rubber covered clamps and proceed in a similar manner until the two segments of the uterus are securely fastened together, when the clamps are removed. In case of removal of pus tubes, I separate the tubes from the broad ligaments and apply the clamps below the tubes, removing them together with the segment of the uterus which comes away. I contend that in all cases where the function of the tubes is destroyed by pus or otherwise, this operation should be done in order to remove the accompanying diseased endometrium.

"The operation is especially applicable in all procidentias after the child-bearing period. In cases of vesicocele, the bladder may be fastened high upon the stump and give most satisfactory results. Another great advantage is that you can retire at night without the apprehension that you may have included a ureter in tying off the uterine arteries. The work can be done in half the time it requires to do the flap operation. In this operation the uterosacral, round and broad ligaments, are drawn so tightly across the pelvis that it makes almost a perfect floor.

"I used this operation in fifteen cases, all with the most gratifying results. The first operation was done June 12th of this year, and the patient seems at the present time perfectly well, with none of those dragging pains in the pelvis and back which so often follow the old operation. In fact, in every case there has been rapid recovery.

"If this operation would meet with the approbation of the profession, and I believe it will, I will take it as a personal favor if they will let me hear from them directly."

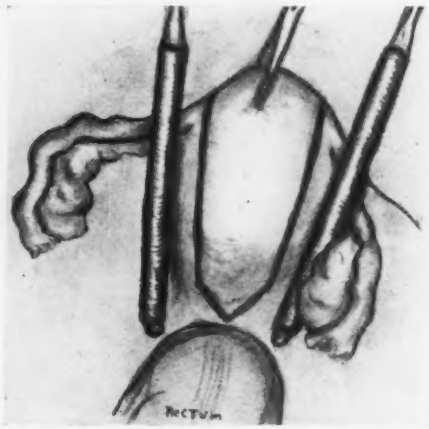

FIG. 5.—Supravaginal hysterectomy where tubes and ovaries are left.

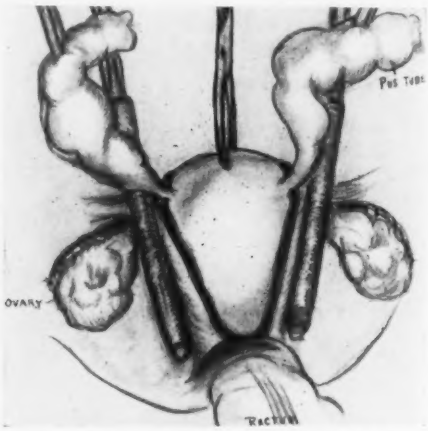

FIG. 6.—Shows method where tubes are removed.

I desire to present the operation, discuss and propose some modifications, in order to extend the principles involved to other procedures. The operation is suitable for a number of conditions where an hysterectomy is indicated. Shropshire proposed it as a supravaginal or subtotal plastic procedure. I have proposed it as a suitable operation for removing the whole glandular part of the uterus and cervix.

I would first like to call attention, rather briefly, to the anatomy of the uterus and its blood and nerve supply. The uterine and ovarian arteries anastomose and richly supply the uterus, and while they anastomose with their fellows of the opposite side, yet the bleeding is not severe when the uterus is cut through the midline anteroposteriorly; nor is there much bleeding when a slab is cut on either side just outside the cavity of the uterus and cervix away from the midline.

The evident and fundamental reasons for the operation are that where, for any reason, the uterus is to be removed without the removal of the ovaries and tubes, the ovaries are left with their blood and nerve supply practically undisturbed. Should the fallopian tubes be diseased and their removal considered desirable, this can easily be accomplished without injuring the blood or nerve supply of the ovaries.

The operation has a very wide field of application in dealing with fibroids of the uterus where a myomectomy is not advisable, and the body of the uterus is to be removed. Shropshire used a specially designed clamp for controlling the blood supply. I have used a straight, rubber covered intestinal clamp on each side so as to better control the bleeding as the slabs are

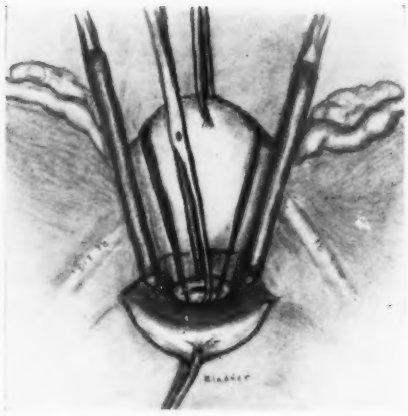

FIG. 7.—Panhysterectomy technic.

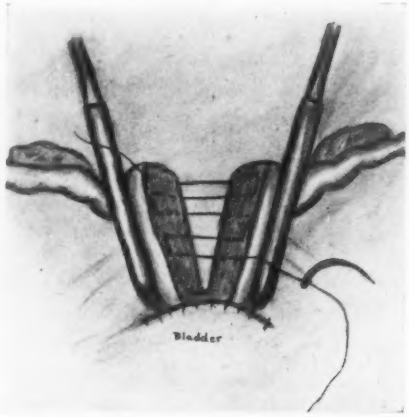

FIG. 8.—Sewing slabs together controlling bleeding.

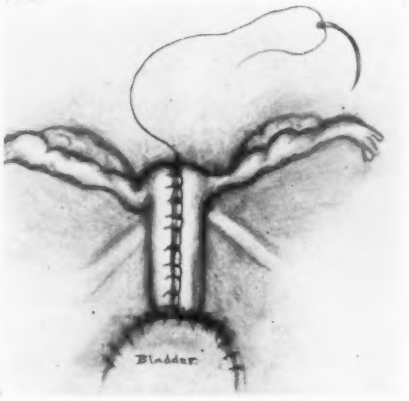

FIG. 9.—Closing serous surfaces.

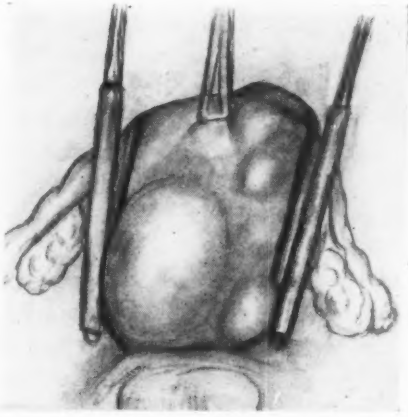

FIG. 10.—Method of removing a fibroid uterus.

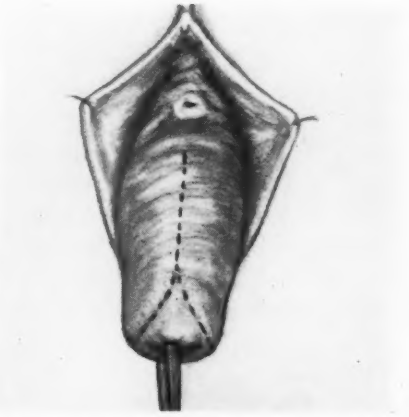

FIG. 11.—Suggested vaginal technic for hysterectomy or for prolapse.

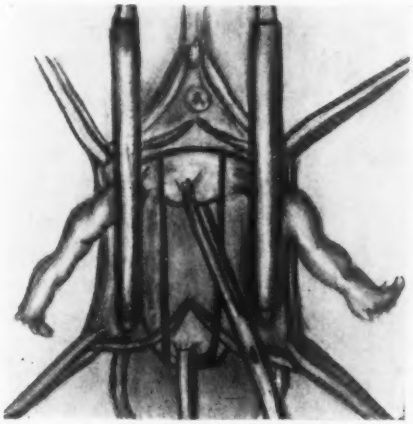

FIG. 12.—Showing method of performing a vaginal Shropshire.

cut. A few vessels may need to be individually ligated. One may use the through-and-through method of the author by threading a long straight needle on either end of a No. 1 chromic catgut or, as I have done in a number of cases, a curved needle may be used as shown in a mattress suture to approximate the plane surfaces of the two slabs. After this the serous or peritoneal borders, anteriorly and posteriorly, are approximated with a No. 1 chromic suture.

I have indicated the abdominal method of performing this operation either supravaginally or where one wishes to remove the gland bearing canal of the cervix also; then the bladder is freed and pushed downward so as to expose the anterior vaginal wall. This is cut through and the cervix is grasped by a volsellum or a toothed forceps and elevated so as to bring it into view. The cut is now extended just outside the cervical canal, and a complete hysterectomy effected, *i.e.*, all of the uterine and cervical canal is removed.

The approximation of the slabs is accomplished by sewing down and around the cervix after approximating the slabs by a quilting or through-and-through suture, as is done in the supravaginal operation. This procedure can be carried out per vaginam in any case where the tumor is small enough to be handled through a vaginal section, as has been done previously. The operation is especially applicable in performing the Chauta-Watkins interposition operation for prolapse of the uterus. The technic is the same as in that procedure except that the whole uterine and cervical canal is removed.

The lateral slabs are handled as in the other procedures. The remaining structures are now closed over by the vaginal flaps, leaving the bladder behind the much reduced mass. Its advantages I think, are self-evident.

In some cases where the prolapse is not too great, and the structures not too much drawn out, the removal of the central part of the fundus and cervix might correct the condition without the interposition operation.

I feel that in preserving all the blood and nerve supply, a firmer union will be obtained between the slabs, and should give better results than performing an hysterectomy in the usual way and then sewing the cut ends of the ligaments together for support, as in the Mayo procedure of hysterectomy for prolapse of the uterus. One not only has better structures for support, but the central gland bearing part of the uterus and cervix has been removed.

The results are excellent in both of these instances—even better than when the whole body of the uterus is left, as in the ordinary Chauta-Watkins or Watkins-Wertheim technic. I have never thought an hysterectomy, by any method, fundamentally sound, as a suitable operation for prolapse, as has been advocated, and have never personally employed it, although it seems to have been successful in many instances.

I trust that the Shropshire operation may merit your careful consideration and trial. It works!

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THE STATUS OF VAGINAL HYSTERECTOMY IN GYNECOLOGIC SURGERY

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A REVIEW of the recent literature on hysterectomy indicates that gynecologists are becoming more radical in removing the diseased uterus in toto. We realize that conservatism is perhaps not always the wisest course in treating a uterus which is producing definite symptoms that experience has proved are very often the forerunners of the now recognized "precancerous" conditions, and eventually of malignant changes. Gynecologists are also beginning to realize that the diseased cervix which is the source of local symptoms such as leukorrhea, pelvic pain or discomfort, and dyspareunia, can also have a marked effect on the general well-being of the patient.

We have been reassured in the last few years by the results obtained in total hysterectomies. A comparison of the mortality and morbidity in series of cases treated by these respective procedures has led to a growing belief that given the necessary skill derived from experience, and the absence of operative hazards above the average, the total operation offers advantages to the patient which overbalance the additional difficulties encountered by the surgeon. In other words, in the hands of the operator lies largely the task of conquering the dangers of total hysterectomy—the longer time needed to accomplish the operation, the greater skill required, increased difficulties in operative technic, and the greater blood loss and danger to vital organs; while the patient derives as advantages—fewer postoperative complications, smoother convalescence, and less fear of disastrous future developments, such as carcinoma of the cervical stump.

In treating conditions which require total extirpation of the uterus, the rule in most of the larger gynecologic clinics throughout the country has been to employ the abdominal route. Vaginal hysterectomy has never been popular; it is a procedure which has waxed and waned in the enthusiasm of gynecologic surgeons, and in the descent of popularity, it has even been frowned upon as an elective procedure. Wayne Babcock[1] inquires: "Is it because it may impose operative difficulties on the surgeon? Or is it that one follows the operation to which he becomes habituated, and finds it inconvenient to select from a variety of methods when a single routine one will serve the purpose?" By way of answering this query, I fully agree with Richardson[4] that: "Complete mastery of a satisfactory technic for total removal of the uterus by both the abdominal and vaginal routes is a minimum standard of operative skill that every gynecologist should exact of himself as early in his career as the circumstances of his period of ap-

prenticeship will permit. . . . For, unfortunately, neither of these operations is best suited to all individuals requiring hysterectomy, and superlative skill in the performance of only one of them becomes but an exhibition of poor surgical judgment and mediocre ability when applied to the correction of pathology that could be better treated by the employment of the other." If we judge that a case requires hysterectomy, our next decision should be what type is best suited to this particular case.

In a series of 206 cases, private and ward, operated upon by me between August 1, 1931, and November 1, 1936, vaginal hysterectomy was selected as the most rational procedure. There was but one operative death, a mortality of 0.48 per cent. Follow up has been obtained on 160 of these cases, in most instances by examination at intervals, and judging from the results obtained, I feel that the choice of vaginal hysterectomy was fully justified. The ages of the patients ranged from 27 to 70 years (Table I). Total abdominal hysterectomy was also performed during this same period 62 times. But the advantages of vaginal hysterectomy far outweigh those of the abdominal route and its correct performance should be the aim of all gynecologic surgeons.

TABLE I

AGE GROUPS—206 CASES

27–35 years	29 cases
36–45 years	100 cases
46–55 years	56 cases
56–65 years	16 cases
66–70 years	5 cases
Total	206 cases

Vaginal hysterectomy yields a lower mortality rate than either subtotal or total hysterectomy. In the present series of 206 cases there was only one death. At the Mayo Clinic, Masson reports that vaginal hysterectomy, when performed on the poor surgical risks for benign conditions, had a mortality of 1.7 per cent in 834 cases, which was 0.2 per cent higher than the 1.5 per cent mortality for 2,085 total abdominal hysterectomies for benign conditions performed upon those considered to be better surgical risks. Many report series of vaginal hysterectomies without mortality. Greenhill[8] reports a mortality for vaginal hysterectomy one-third of that for abdominal. Heaney[7] cites 695 vaginal hysterectomies with but three deaths, and Wayne Babcock, 300 with no deaths.

The acid test of the worth of a procedure is, however, the degree of morbidity it entails, and I think there is no one who will dispute that vaginal hysterectomy primarily submits the patient to less surgical shock than abdominal hysterectomy, and that the convalescence is thereby bound to be less stormy with fewer complications and less peril of infection. The average number of days of hospitalization for the present series was 12, which in itself is indicative of a smooth postoperative period. Follow up and

examination at varying intervals of 160 of the 206 cases reveal a very satisfactory result: Complete relief of symptoms in all but one case, only slight bladder symptoms remaining in two cases, dyspareunia in three cases, and slight vaginal discharge in six cases. When the symptoms of which these patients complained prior to operation are analyzed, the end-results are an encouraging testimonial to the value of vaginal hysterectomy in gynecologic surgery (Table II).

TABLE II

SYMPTOMS—206 CASES

	No. of Cases
Leukorrhea	202
Backache	205
Urinary symptoms (incontinence, frequency, pain)	108
Vaginal bleeding, postmenopausal	24
All other types	161
Pelvic pain	79

Another advantage is the rapidity with which the vaginal removal of the uterus can be accomplished, in conjunction also with perineorrhaphy, repair of the cystocele, correction of obstetric injuries, and other surgery. The average operative time in the present series was 40 minutes (Table III).

TABLE III

OPERATIONS

Vaginal hysterectomy	20
Vaginal hysterectomy and perineorrhaphy	186
Complete tear operation	2
Salpingo-oophorectomy	7

In this series, vaginal hysterectomy was chosen: (1) To extirpate a diseased uterus and cervix; and (2) As an incidental procedure to correct a complete prolapse of the uterus and vagina.

I believe, in dealing with a diseased uterus and its more diseased cervix —conditions which are usually accompanied by menstrual disturbances and pelvic pain—that more radical therapy than we have employed in the past is imperative. Our hope that the endocrines would solve our difficulties in controlling bleeding from an otherwise healthy uterus has not been realized, and our greater fear of the development of malignancy in a diseased uterus has led us to the adoption of this more radical procedure. If we can prepare our patients and master a technic which reduces the mortality and morbidity to a negligible factor, then we should advocate the performance of hysterectomy more often, and in the absence of extensive inflammatory conditions of the pelvis and very large fibroids, we should usually perform the operation through the vagina.

The first indication for which vaginal hysterectomy was performed in this series, *i.e.*, a diseased uterus, included cases of fibroids not larger than a three months' pregnancy, early malignancies of the cervix and corpus,

fibrosis of the uterus, menopausal bleeding, all associated with a very badly diseased cervix. In all these cases there was, of course, a certain amount of prolapse, cystocele, and relaxation of the pelvic floor. It would seem that in women at or near the menopause, complaining of pelvic pain, vaginal bleeding and discharge, with an enlarged fibrotic uterus, chronically diseased cervix, cystocele, and lacerated perineum, where a fair degree of prolapse exists, that a total removal of the uterus from below with correction of the cystocele and rectocele, is a logical and indicated procedure.

Former treatment of chronic cervicitis by local measures—cauterization, partial amputation, diathermy, and more recently conization—has often not given the patient the relief she desires. While I do not attempt to discredit these forms of treatment for the diseased cervix when the pathologic changes are confined to the cervix, or when the symptoms are strictly local, however, when the infection has extended beyond the cervix, and when the symptoms point to disease also in the body of the uterus, and the parametrium, or when the condition is producing systemic changes such as loss of weight or anemia, and is a definite focus of infection, I believe that the more radical treatment is indicated. Conservative plastic operations, cauterizations, and conizations often result in stenosis of the cervix, which is the cause of changes leading to a train of symptoms eventually requiring extirpation of the entire uterus (Table IV).

TABLE IV

PATHOLOGIC CONDITIONS FOR WHICH VAGINAL HYSTERECTOMY WAS PERFORMED

CERVIX	No. of Cases	UTERUS	No. of Cases
Diseased cervix	187	Fibroids	38
Lacerations	138	Fibrosis	148
Erosions	46	Pyometra	4
Stenosis	19	Atrophied	16
Polyp	5	Procidentia	43
Suspected malignancy	17	Cystocele	190
Previous amputation	8	Lacerated perineum or rectocele	206

In extensive cystocele associated with procidentia, repair of the cystocele by replacing the uterus and reconstructing the fascial supports will not suffice, as the weight of the uterus will often cause a recurrence. We must not lose sight of the fact that in procidentia the condition is not altogether due to the failure of anterior supports of anterior fascia, but to the failure of posterior supports as well. Therefore, some form of shortening the entire broad ligament is necessary, and not merely shortening of one or more of its component parts. This can be effected only by shortening the broad ligaments at their midportion, which necessitates removal of the uterus, be it diseased or not. This step enables us to shorten the broad ligament and thereby construct a satisfactory sling or support for the replaced bladder. In the great majority of cases of procidentia, the patient has long passed the period when the uterus is a functioning organ, and

therefore its removal is only an incidental procedure, but one that is necessary if we are properly to correct this always distressing condition.

In this series there was only one case in which the operation did not bring about the desired results, and which, therefore, might be considered as a failure. In all the others the patients evidenced satisfaction at the general improvement in health and in the cure of distressing symptoms.

CONCLUSION

I think, therefore, that vaginal hysterectomy has a valuable place in gynecologic surgery as a procedure offering many advantages, and believe that an analytic consideration of its usefulness has often been overlooked in the past. I am of the opinion that there are, at times, definite indications for its election over any other procedure, and am further of the opinion that the results obtained in these selected cases offer adequate and convincing testimony in support of this conclusion.

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DISCUSSION OF THE PAPERS OF DOCTORS BURCH, MILLER, MOORE, AND TYRONE

DR. JOHN W. PRICE, JR. (Louisville, Ky.).—This is a very interesting group of gynecologic papers. Relative to Doctor Burch's paper on perineorrhaphy, I think he has developed an excellent operation and satisfactorily demonstrated how he dissected the fascia and sutured it properly. The operation we perform is not complicated by the variety of sutures he showed. We do what I have always chosen to regard as a modification of the Mayo flap splitting operation. We have been using it for a good many years. The essential step is to expose the levator ani and employ a continuous button hole suture, which practically always controls any hemorrhage. It has been noted that if one does not enter the fascia of the levator ani, bleeding seldom occurs.

Regarding Doctor Moore's type of hysterectomy, which I would regard as a subtotal hysterectomy on account of the large amount of the wall of the uterus he leaves behind, the only criticism I would offer is that in cases of fibroid tumor of the uterus it would be possible that some small fibroid which was unseen at the operation would remain in the wall and would subsequently grow and have to be removed. In my work I have been un-

fortunate enough to have fibroids develop in the remaining stump of the cervix following subtotal hysterectomy, and I have operated upon some cases for the same condition where the patient had been previously operated upon in other clinics. In my own practice the incidence of fibroid occurring in the stump of the cervix has not been frequent but other men have reported these cases in considerable numbers.

Regarding Doctor Tyrone's advocacy of vaginal hysterectomy, I think he is correct in stating that these patients do have less shock. I have limited vaginal hysterectomy to that group in which I thought it was particularly indicated, using it rather more in elderly women. Formerly women in the neighborhood of 40 were included in this category, but I think the age should be raised to 50 or 60 to make the vaginal type proper for "elderly" women. I do not like to perform a vaginal hysterectomy if I think there are many adhesions in the abdomen. In the group we have in the City Hospital, with the complications of cystic ovaries, *etc.*, I prefer going in supravaginally. In the cases with complete prolapse where the uterus slips around like a ball on the end of a string I think a more rapid operation such as the one Doctor Tyrone described is more desirable.

Dr. Robert L. Payne (Norfolk, Va.).—I enjoyed Doctor Burch's presentation of the operation he has developed for perineorrhaphy, and while I presume that support must be provided, I think the real promise is that we are dealing with a definite hernia in the rectocele. I do not think that the separation of the levator ani on the sides only, as shown in Doctor Burch's drawings and technic, is quite sufficient. I think the rectocele must be freed in its entirety, just like an hernial sac, all around, and when you have done that you can reduce the sac and push it down and you will have more levator ani for closure, which does not obtain when the other procedure is carried out with only the lateral sides of the rectocele freed from the levator ani.

To demonstrate what I mean: Instead of freeing the levator ani only in a vertical way and trying to bring that across, it should be freed all around and the rectocele reduced, which will leave at least half an inch more of the levator ani for closure over the rectocele. (Ref.: J.A.M.A., 78, 574, February 25, 1922.)

Doctor Tyrone's paper interests me immensely. I congratulate him upon his splendid results, but there is a complication of vaginal hysterectomy—namely, genital prolapse—that he did not touch upon, which I consider very important. When vaginal hysterectomy is performed by a competent surgeon, prolapse of the bladder and the vagina afterwards is a very rare occurrence. Frequently, however, this operation is performed by an incompetent operator, and then complete genital prolapse of the bladder and vagina, not infrequently, takes place. I encountered this unusual condition several years ago, and could find no operative method of handling the problem in the literature. The problem was apparently solved by a method I developed, which was published in the Arch. Surg., 20, 637, April, 1930. We successfully have operated upon four cases with this condition. Fourteen other instances, which have been operated upon by other surgeons, employing the same technic, have been reported to me, with successful results being obtained in each case. It is of course highly essential that a very snug perineal repair be effected in addition to the intra-abdominal technic.

Dr. R. H. Jackson (Madison, Wis.).—I wish to thank Doctor Moore for his explanation of the smoothness with which patients convalesce after this type of operation. The name which I have used to designate it is sub-

cortical vaginal hysterectomy. My technic is a little different from that described by Doctor Moore, but achieves the same end, so that crushing injury to the broad ligaments is avoided. After the fundus uteri is freely mobilized in the vagina, a tenaculum is applied at a point just below the summit of the fundus on its posterior surface, another tenaculum is applied on the anterior surface of the fundus an inch below its summit. Rubber covered bayonet clamps are applied to the broad ligaments sufficiently tightly to control the arterial supply (not crush the tissues). With the scalpel or radio knife, two incisions are started just below the upper tenaculum and pass in concave curves over the fundus to just within the attachment of the round ligaments, and thence turn slightly downward and meet at a point behind the cervical os. With careful deepening of these incisions from above downward, leaving a uterine shell approximately one-eighth inch in thickness, the uterine body with its mucous membrane cavity intact is removed, save for the above mentioned cortex or shell. All visible vessel ends are ligated, the control forceps unlocked, and all bleeding points secured. A single No. 2 chromic catgut suture is placed at the site of the tenaculum —the ends being left ten inches long to serve as the anchoring "crown suture" under the pelvic arch. The uterine shell is then reduced to approximately the size and shape of a finger by interrupted or spiral sutures and then anchored by the "crown suture." The lateral reduction gained by thus approximating the points of origin of the round ligaments tightens the broad ligaments very satisfactorily, and this, together with the "crown suture" and the insertion of a uterosacral ligament suture at the opposie end, insures an excellent pelvic floor. The operation is completed in the usual way by overlapping the inelastic layer of fascia as in the Mayo umbilical hernia operation. In my experience with this operation during the past 25 years, it presents certain advantageous features:

(1) There are no raw surfaces left in the peritoneal cavity.

(2) There is less danger of thrombosis and possible embolism.

(3) There is less danger of sloughing in the vaginal vault which sometimes accompanies the multiligatured and sutured, crushing clamp-broad ligament procedure.

(4) In well over 100 such operations, there has been no known instance of recurrence of cystocele or prolapse.

(5) Convalescence is definitely smoother, due possibly to the gentler handling of the tissues and the avoidance of injury to the nerve structures, as pointed out by Doctor Moore in the Shropshire operation.

(6) Another advantage, possibly a small one, but psychologically of value, is that of being able to tell the patients that all of the womb was not removed. They feel less unsexed.

Naturally one would not use this method in a uterus well peppered with small fibroids on the posterior fundal wall. Preferably, it is used in elderly patients with prolapse and cystocele, the remaining cortical uterine body being used as in the Watkins' interposition operation.

Dr. William T. Black (Memphis, Tenn.).—The technic Doctor Burch has given us is splendid. However, I think in operating we all use different types of technic in nearly every perineorrhaphy. We cannot follow the same one in all cases. It is necessary to have a very free dissection. I have seen apparently perfect perineorrhaphies in women, who had lateral lacerations of the vagina, that had to be reoperated upon.

I think we should thank Doctor Miller for presenting the subject of

echinococcal disease of the uterus so that we may look out for such cases in the future.

Doctor Moore's exposition seems to me to be more of an incomplete rather than a complete supravaginal hysterectomy. It may be all right, but I believe it better to remove the entire body of the uterus. If we save uterine tissue, it is better, I think, to save the endometrial portion of the uterus, so as to preserve menstruation and prevent psychoses. However, I do not advise this in the usual case requiring a supravaginal hysterectomy.

In my opinion, Doctor Tyrone is correct in saying that vaginal hysterectomy is an overlooked operative procedure. Certainly for those patients who have indications for such an operation, it is ideal. It is particularly suited to elderly women who are obese. You can perform it under local anesthesia (if necessary) and there are practically no postoperative complications. I think in fibroid tumors of any size the supravaginal route is the best. In 57 per cent of the cases of fibroids noted in the records of John Gaston Hospital, chronic inflammatory pelvic disease was concomitant, which would preclude a vaginal hysterectomy. Certainly we should have complete vaginal relaxation. It is suitable in cases with a large cystocele and rectocele accompanied by procidentia. In cases of vaginal prolapse following a vaginal hysterectomy referred to by the speaker, fixation of the vagina to the abdominal wall, in rare cases, gives a splendid result. I described such a case several years ago, which was abstracted and published in the Year Book of Obstetrics and Gynecology, but have not had an occasion to use the method often since. If one will take pains in bringing the round and broad ligaments, and also the sacro-uterine ligaments together, you get a very good support. Permanent sutures to the pubic fascia from the above structures and taking pains in grasping the para-uterine and paravaginal fascia will usually prevent a vaginal hernia.

Dr. C. J. Hunt (Kansas City, Mo.).—I wish to say a few words in behalf of the value of vaginal hysterectomy for a selected group of cases who are past the menopause or advanced in years and have a grade three or four prolapse of the uterus accompanied by cystocele and rectocele. Vaginal hysterectomy in this group of cases eliminates the prolapsed uterus, prevents a possible future malignancy of it, and affords ample approach for the repair of the cystocele.

Two cardinal principles are essential in the operation of vaginal hysterectomy for uterine prolapse with cystocele. In the repair of the pelvic floor after hysterectomy, the broad ligaments are sutured together in the midline; and on each side of the urethra they are securely anchored to the pubic fascia. This prevents future prolapse of the bladder over the broad ligaments. In addition, it is necessary to obliterate the cul-de-sac by a suture which picks up along with the uterosacral ligaments the peritoneum on each side and between. This prevents future culdesac herniation.

In the repair, one must keep in mind the anatomy of the pelvic floor and remember that the prolapsed condition is simply that of herniation through faulty support. The pelvic diaphragm is chiefly made up of the levator ani muscle and the pelvic fascia. The muscle converges from its origin along the under surface of the pubic rami, obturator fascia and ischial spine toward the midline uniting in a median raphé. The fascia is very definite and at points where support is needed it becomes thicker and stronger, as illustrated by the vesicovaginal fascia, the fascia around the hiatus uteri and the rectovaginal fascia. When the continuity of these structures is broken there is an herniation of the organs they support; therefore, a repair of vesicocele

consists in the repair of the fascia through which the bladder has herniated.

The same applies to the reconstruction of the vaginal floor. The uterus prolapses when the supporting structures have been weakened and when it assumes the retroverted position. The uterus to prolapse must be in the same axis as the vagina, a retroverted position. Intra-abdominal pressure and posture then aid in the descent. When the anterior position of the uterus is maintained the antiflexion is increased by intra-abdominal pressure and there often occurs an elongation of the cervix. The effects are in direct relation to the degree of pelvic floor defect and the anatomic position of the uterus.

I have been especially interested in repair of pelvic floor defects and have followed very closely the methods and principles so well emphasized by Dr. J. C. Masson. The results in the type of case that I have discussed have been very satisfactory.

Dr. F. Webb Griffith (Asheville, N. C.).—I would like to ask Doctor Moore to tell us a little more about his vaginal hysterectomy. Wherein is it superior to the Mayo operation? In short, what does he hope to accomplish that the Mayo operation will not do?

I was interested in Docter Tyrone's paper, for I am sure we are not doing enough vaginal hysterectomies. One reason is that most of the gynecology in the smaller towns is done by general surgeons who have not had special training in gynecology, and frequently they do not know how to perform a vaginal hysterectomy safely and satisfactorily. That, however, is not the only reason. It was my privilege to spend nearly five years on the gynecologic staff of one of our well known hospitals. In that period I am sure I did not see over two dozen vaginal hysterectomies. It is true that was 25 years ago, but I understand that the ratio of vaginal to abdominal hysterectomies in that institution is still very low. Possibly the explanation is since the gynecologists there do such excellent "abdominal gynecology," they do not often feel the need of doing an hysterectomy per vaginam.

Regarding Doctor Payne's remarks, I do not see how you can get much worse cystoceles than sometimes follow an abdominal hysterectomy for marked uterine prolapse. There is no stereotyped operation for these distressing cystoceles. A rather extensive denudation of the vaginal portion of the bladder and closing over as in the ordinary cystocele operation, followed by a thorough perineorrhaphy, almost to the occlusion of the vagina, offers the best prospect for cure, or at least of benefit. It may, or may not, be necessary to follow that by laparotomy, tacking up the bladder to the abdominal peritoneum, and also placing sutures here and there to any of the remaining pelvic structures, which might help to hold up the bladder.

Dr. Joseph E. J. King (New York N. Y.).—I have enjoyed these papers very much, but I had hoped that Doctor Miller's consideration of echinococcal disease in general would offer an opportunity in the discussion for someone to say something about extirpation of echinococcic cysts of the liver rather than marsupialization.

In 1926, I reported a case of complete extirpation of an echinococcic cyst of the liver. It projected from the lower border of the liver, was about the shape and size of an eggplant, and was surrounded by a considerable amount of fibrous tissue. This permitted complete extirpation, leaving a horseshoe-shaped defect in the lower portion of the liver. It was quite simple and easy, and the patient made an uneventful recovery.

This contrasted markedly with two other cases which I have observed,

and in which marsupialization was employed. These wounds persist for a long time and are dirty and slow in healing. Complete extirpation, when this can be effected, is advised.

Dr. John C. Burch (closing).—Each rectocele must be treated according to its own particular needs. The method of dissection described in the paper permits of this individualization.

Dr. Curtis H. Tyrone (closing).—I wish to thank the Fellows for their discussion. I did not mention drainage, but when I first began the work I drained some of the cases and found no difference in convalescence in the cases that were drained and those that were not.

As to the problem of prolapse of the vagina that Doctor Payne brought up, I have seen that following every type of hysterectomy, and it is a very distressing condition. This condition has not developed in any case that follow ups were obtained on in this series of cases.

I can recall assisting the late Dr. C. Jeff Miller in operating upon several cases of vaginal prolapse following hysterectomy done elsewhere, and he always repaired the prolapse by a vaginal operation with excellent results.

The reason I am advocating vaginal hysterectomy is because one can correct the obstetric injuries and better prevent the development of this condition than by abdominal hysterectomy.

Dr. John T. Moore (closing).—I wish to thank Doctor Jackson for giving me a little help. It looked as if I nearly batted out on this operation of Doctor Shropshire's. It would take me too long to say everything that has come into my mind to say while this discussion has been going on. I have not touched on many of the things that have been brought out in the discussion. I admit that all lacerations should be repaired when performing this operation. Every woman should be completely operated upon, if possible. We keep in mind, however, the so called "shot gun" treatment, the basis of which is that if you do not do any good for the patient you should do no harm. If one reads the literature I think he will come to my conclusion, that the uterus has more function than that of bearing children. I am not wedded to any particular treatment. This is not my baby, it is a baby I have adopted, but I think there is more virtue in it than has been accorded it. One important thing is that with the average clamp applied to the tissues and a ligature placed on the vessels you have danger of a thrombus. If you can get away from that danger it would be well to do so. You understand these slabs can be cut away without much hemorrhage, and the blood and nerve supply are but little disturbed. A supravaginal hysterectomy is easier to perform than a total hysterectomy, and I believe that the difference in mortality in "occasional surgery" will overcome the ½ to 1 per cent of cancer shown to occur in the cervices left behind. This technic will reduce the death rate by the usual procedure of hysterectomy. So I believe a supravaginal hysterectomy will leave a higher percentage of patients alive and well than will the complete hysterectomy. It is better for the surgeon to select his cases. I do not perform a complete hysterectomy all the time; sometimes I prefer a vaginal hysterectomy. I try to have an appreciation of the danger of carcinoma in all cases. I can perform a complete hysterectomy better by this method than I can by the other. If you are not too wedded to any particular type of operation, I think you will try this procedure and will find the results are better. In employing it you do not interrupt any important nerve trunks—only the small twigs. In this plastic method you conserve the complete blood supply

to the ovary and the slabs. That is, you have preserved the most essential circulation. The idea of leaving a part of the uterus in which a fibroid may develop is possible, I admit. But I have a sense of touch which I think will detect any fibroid as small as a mustard seed. All of us can cultivate that type of touch if we try. Good strategy is to always leave yourself where you can get out if the other fellow licks you, and it is a good plan to perform an operation in such away that you can change the type of procedure if it is necessary.

Doctor Payne spoke of a fixation operation. I was talking about the interposition operation—the Chauta-Watkins type. I would not advocate using this type altogether in procidentia, but I think you can get a better result from this modified procedure than by any other I know of. I have had occasion to look up every technic that is mentioned anywhere. Doctor Shropshire mentioned the preservation of the nerve supply complete in his procedure, and very little interference with the blood supply. If you have a procidentia to deal with you must consider the case and adopt the technic most suitable for that case. I have seen people perform hysterectomies where the whole bladder and uterus were coming down and even out. I admit the excellent work of Dr. Charles H. Mayo, and with all the vessels tied and the structures brought close together you may get a very good result, but not so good as you do with these slabs left as a support, with part of the uterus left behind, with their complete blood and nerve supply to the ovaries. Their function is carried on by giving their usual endocrine supply. I accept this interposition operation as a standard method, in the hands of the best gynecologists in the country, of curing severe prolapse. However, I think it is better to remove all the endometrium of the fundus and of the cervix, sewing these slabs together and leaving a smaller body interposed. Doctor Jackson illustrated this but in a different technic. The body of the uterus is not sewed to the abdominal wall but puts the bladder behind the uterus. The best type of repair must be given in each case. In my first paper before this Association I presented a method of curing prolapses of the uterus. Doctor Watkins of Chicago thought his interposition operation solved the problem in all these cases. I think the Chauta-Watkins operation a good one in suitable cases, but I use other methods at times.

I like the technic Doctor Burch has shown us and I believe he will get good results. It is an excellent operation, but a man who hunts squirrels in the woods where there are lions and tigers must carry not only a squirrel gun but one for lions and tigers.

A SATISFACTORY METHOD OF REPAIRING CRUCIAL LIGAMENTS

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RUPTURE of the crucial ligaments is a severe injury, and with the increasing number of automobile, industrial, and football injuries, is becoming more frequent. Rupture of the posterior crucial ligament is probably more rare than rupture of the anterior.

If, after an injury to the knee joint, the tibia can be displaced backward or forward or rotated inward in the extended position, injury to the crucial ligaments is diagnosed. If, in the extended position, the tibia cannot be displaced forward, the anterior crucial ligament is not ruptured. If, in full flexion, the tibia cannot be displaced backward, the posterior crucial ligament is not ruptured, or, in other words, in anterior displacement of the tibia with the knee extended, you have a rupture of the anterior crucial ligament, and if the tibia can be displaced backward with the knee flexed, the posterior crucial ligament is ruptured.

All of these crucial ligament injuries should first be treated by prolonged immobilization of the knee joint—three or four months—and the conservative treatment given a fair trial before operation is resorted to.

Suture of the ruptured crucial ligaments is a rather difficult procedure, and it is practically always necessary to resort to some type of reconstruction operation to repair them. Such operations have been devised and reported by V. Putti, Haygroves, Alwyn Smith and others.

I wish to present an operation which has given good results in my hands, and which I feel will work equally well in either rupture of the anterior or posterior crucial ligament. Some of the cases which I have seen have a relaxation of the capsule of the knee joint, and, in my opinion, the capsule of the knee joint should always be tightened anteriorly at the time of the operation. I prefer to use the fascia lata in repair of the crucial ligaments, and feel that a very liberal strip of fascia should be used so that when the fascia lata is folded or rolled together, it should be the size of a lead pencil or the little finger in order to give greater strength. I also believe that the strip of fascia lata should be long enough to pass through the joint and out below the capsule, then externally over the capsule, to be fastened finally to the site of entrance of the fascia lata in the femur, thus forming a complete loop and further strengthening the joint.

I believe also that the fascia lata should enter the femur above the condyle instead of through the condyle, also that the fascia lata should enter the knee joint in the center of the intercondylar notch, and pass through the tibia just anterior to the tibial spines, and make its exit from the tibia just

anterior to, and below, the head and neck of the fibula. The above points, in my opinion, are very important, and if they are followed, the operation described below will give good results.

OPERATIVE TECHNIC.—A long incision is made over the external surface of the thigh, extending from just above the knee joint involved well up on the thigh to allow for a good long liberal strip of fascia lata (Figs. 1 and 2). The defect in the fascia lata caused by removing this strip of fascia is then closed with chromic gut No. 2. This leaves the lower end of the fascial strip free at the upper end and still attached near the knee joint (Fig. 2). The knee joint is then opened through a split patellar incision and inspected (Figs. 3 and 4). The muscles above the external condyle of the femur are then separated by blunt dissection down to the bone, held aside by blunt

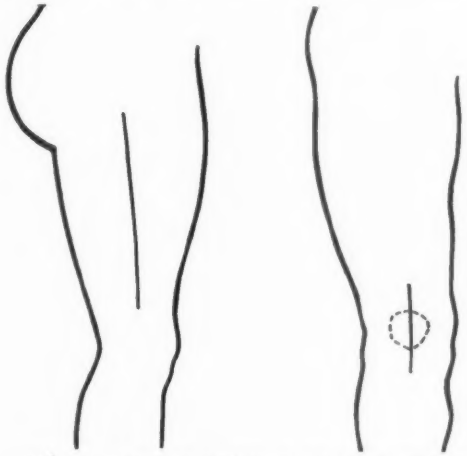


FIG. 1.—Shows the position of the lateral incision on the thigh for the removal of fascia lata, also the location of the split patella incision.

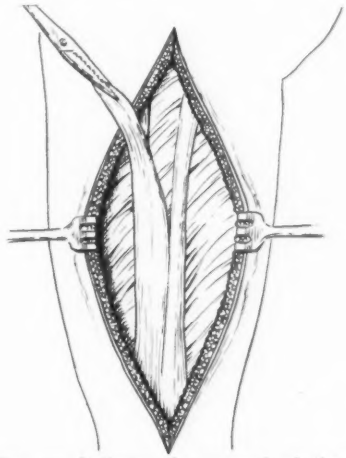


FIG. 2.—Indicates the removal of the strip of fascia lata.

retractors, and a drill about the size of a lead pencil is passed through the femur from a point above the external condyle of the femur to the middle of the intercondylar notch. Then, with the knee flexed to a right-angle and the knee joint open, the same drill is passed from a joint just anterior to the tibial spines in the direction of a point on the tibia just below the capsule of the knee and slightly anterior to the neck and head of the fibula. In some instances where it is not possible to obtain the proper angle with the drill from above, it is necessary to make the openings communicate in the tibia below through another drill hole in the position mentioned (Figs. 5 and 6).

We are now ready to pass the strip of fascia through the holes in the femur and tibia. To do this, I prefer a suitable length of phospho-bronze wire doubled on itself, the strip of fascia being passed through the looped end. The strip of fascia is now pulled through the knee joint fairly snugly, with the knee extended. The split patellar incision is now closed, tightening the joint capsule as much as is thought necessary. If there is much relaxation, it may be necessary to remove a small portion of the patella. The capsule

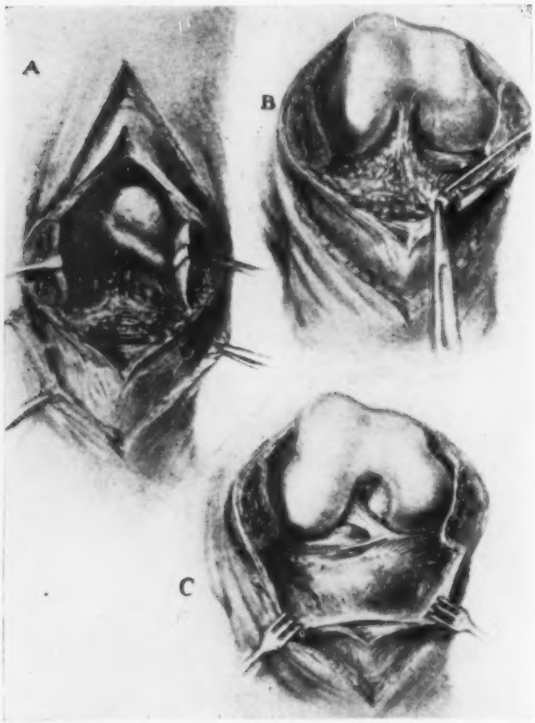


FIG. 3.—Demonstrates the split patella incision, with the exposure of crucial ligaments and condyles of the femur.

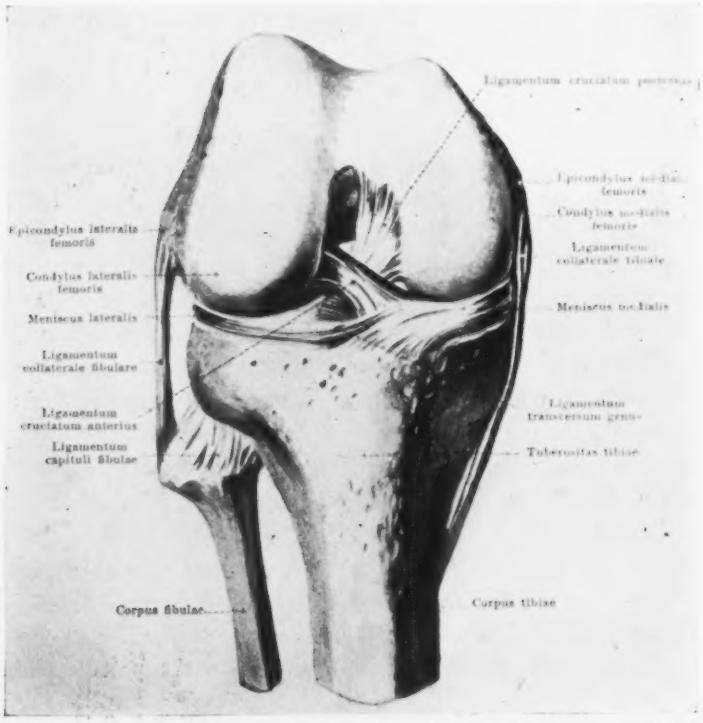


FIG. 4.—An anatomic cut demonstrating the crucial and other ligaments of the knee joint.

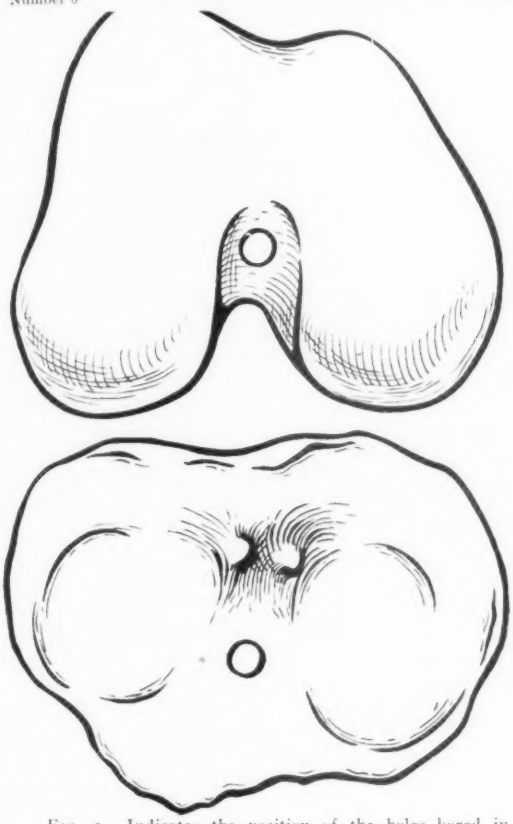


FIG. 5.—Indicates the position of the holes bored in the fibula and tibia as near the center of the joint as possible.

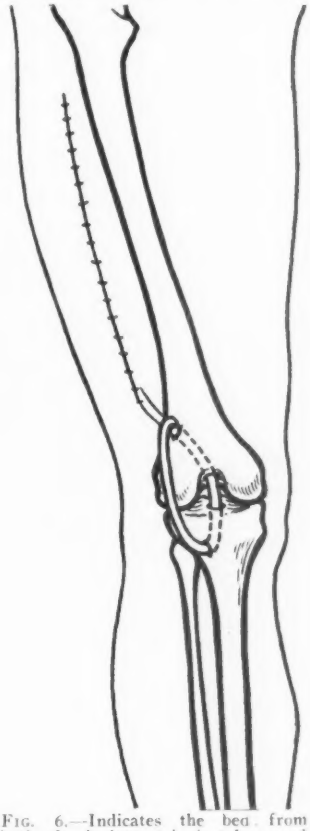
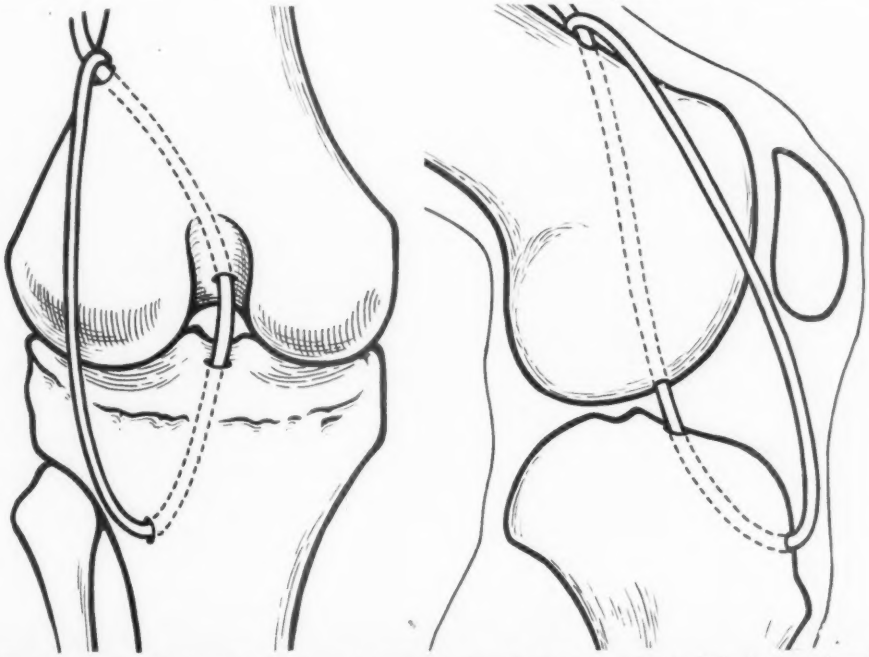


FIG. 6.—Indicates the bed from which the fascia lata strip is taken, and the manner in which it is passed through the knee joint.



FIGS. 7 and 8.—Demonstrate the fascia lata passed through the knee joint in both the lateral and anteroposterior positions.

of the knee joint and patella are sutured with interrupted chromic gut No. 2, the knee joint being closed. The strip of fascia is now passed externally over the joint capsule, lateral to the patella, and fastened to itself at the point where the fascia entered the femur above the external condyle (Figs. 7 and 8). The strip of fascia is also sutured to the capsule of the knee joint at intervals with interrupted sutures, all of this being done with the knee extended and the fascial strip pulled fairly taut. The skin wounds are now closed with fine chromic gut, and sterile dressings applied. To maintain extension of the knee joint, we use either a circular plaster of Paris splint from hip to toe, bivalved, or a posterior plaster shell. This splint is worn six weeks. After this time, the patient is fitted with a simple leg brace fastened into the shoe, with no motion at the ankle joint and a lock joint at the knee, and permitted to walk. After eight weeks, gradual motion is started in the knee joint with gentle massage. In about six months from the date of the operation, these patients have a good functioning, serviceable joint.

THE EFFECTS ON BONE OF THE PRESENCE OF METALS; BASED UPON ELECTROLYSIS

AN EXPERIMENTAL STUDY

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THE reasons for undertaking these experiments on the reaction of bone to metals were, in the first place, the fact that extremely variable bone changes and clinical end-results have been observed when various metals recommended by research workers or by instrument houses have been used indiscriminately in the fixation of fractures. In the second place, we found much confusion in the reports of those who have performed exhaustive experiments to determine which metal or combination of metals was the most consistently tolerated by bone. Moreover, the directly contradictory opinions, and totally variable findings, of previous experiments gave us no clue as to the ideal metal or combination of metals for bone repair, upon which complete dependence could be placed.

Doisy,[4] in 1894, experimented with the then new metal, aluminum, and found that it produced irritation of the bone like any other foreign body. He concluded, however, that its salts were of weak toxicity and therefore that the metal was safe to use.

LeFort,[13] in 1918, observed that the body tissues reacted differently to bullets of different metals in spite of the absence of infection or other extraneous cases. He felt that the variations could possibly be explained by the action of the different metals used in alloys.

Algave[1] used silver wires in his experiments and stated that he was able to produce a chloride of silver which was harmful to the repair of bone.

Hey-Groves[8] claimed that nickel-plated steel did not produce any irritating effects on the tissues, and that magnesium produced destruction of bone if it was in contact with the cortex alone instead of in the medullary cavity. These experiments were quite rudimentary and were not checked by microscopic examination.

Rugh[19] experimented with 16 different types of metal, of which tiny pieces were placed in cultures of *Staphylococcus aureus* and *B. pyocyaneus*. He found that iron, steel, copper, zinc, and nickel, which are readily oxidized by body fluids, frequently cause aseptic suppuration. Silver, gold, and tin were unaffected by the body fluids.

Zierold[21] operated upon a number of dogs in which he drilled holes in numerous bones in various parts of the body and placed pieces of these metals in the holes: Gold, silver, aluminum, zinc, lead, copper, nickel, carbon, steel, stellite, *etc.* The tissues about the pieces of copper showed much dis-

coloration and marked overgrowth of the bone. About the gold, silver, and aluminum there was excessive subperiosteal growth of bone. Lead caused indifferent bone reaction. Nickel produced marked irritation and some stimulation of new bone. Iron and steel caused discoloration and soft tissue reaction. Gold, aluminum, and stellite were readily tolerated by bone and became encapsulated early. Silver and lead, which are easily corroded, caused more connective tissue reaction. Zinc was corroded readily and interfered markedly with bone regeneration. Zierold concluded that steel and iron definitely inhibit bone regeneration and that stellite causes the least reaction of all the metals used. (Stellite is an alloy containing 58 per cent cobalt, 35 per cent chromium, 4 per cent tungsten, plus small amounts of iron, carbon, *etc.*).

Trout[20] inserted steel screws in the bones of young rabbits and noted an arrest of growth of bone.

Leriche and Policard,[16] in their elaborate studies of the physiology of bone, found constant necrosis of bone under the metal plates which were used to immobilize fractures. After excluding injury, pressure, and infection as possible causes, they still found evidence of destruction which they assumed was due to the chemical nature of the fluids about the metals.

Jones and Lieberman,[9] in a recent thorough study, have shown that varying reactions of bone to metals result from the use of impure metals or from alloys of unknown composition. They experimented with small tacks of various types of rustless steels, which were placed in holes in the femurs of dogs. Five dogs were used. As the tacks were removed they were weighed to compare their weights before and after the experiments. Tacks of the same metals used in the dogs were soaked in Ringer's solution and kept at body temperature for 30 days to serve as controls. It was observed that there are many alloys in use which vary notably in chemical composition and that it is necessary to understand their composition to get the best clinical results. This is especially true of the new alloys known as rustless steel. It was suggested that no metal should be used in bone which is corroded rapidly by immersion in Ringer's solution. It was noted that there was much reaction in the soft tissues about all the screws which were used. Such soft tissue reaction seemed to be related in some way to metallic corrosion. The authors decided that chrome-nickel rustless steel was the best metal to use in bone, since corrosion of this metal was minimal. However, this alloy did irritate the bone somewhat.

Only an occasional observer has mentioned the possibility that electrolytic reactions between various metals used in bone repair surgery might be an explanation for the interference with bone growth which occurs in the presence of metallic fixation devices. A few French experimenters have proposed this idea, although they have failed to substantiate their inferences by careful chemical studies. Rolland,[18] for instance, experimented with silver, bronze, iron, and galvanized iron in bone and believed that the plates of metal were eroded by electrolytic action. However, he did not attempt to prove that this

explained the toxic effect of metals on the healing bone. Le Grand[14] repaired a fractured radius with a plate of steel and two circular wires. An excess of callus formed, and he wondered if the presence of these two metals bathed by the organic fluids of the body might not have formed a true electric couple which irritated the bone. He later performed several experiments in rabbits using copper, iron, and zinc, and even though there were few experiments, and these superficial, there was strong evidence that, with the two different metals in contact with bone, certain phenomena were produced which interfered with bone growth. Zierold,[21] whose work has been referred to before, suggested that the soft tissue reactions were mainly dependent upon the disintegration of the metals used. He did not, however, suspect electrolytic reaction. Galfre[7] assumed that electrolytic couples were to blame for all complications in the use of metal appliances in fracture work. He wondered if some of the unfavorable tissue destruction might not be due to the reaction of the metal and the calcium of the bone itself. He then erroneously concluded that the action of bone upon metal and the metal upon periosteum was the cause.

Cretin and Pouyanne[2, 3] attempted to explain the cause of variable reactions of metals on bone on a basis of cellular stimulation. They, therefore, were careful to experiment on normal bone with a minimum of trauma. They also studied all their specimens microscopically. Since the conclusions previously reported were so contradictory, they felt that much obscurity had been introduced into this problem. They operated upon guinea-pigs and placed metals such as aluminum, silver, copper, iron, magnesium, nickel, lead, and zinc in the bones. First they tried to see what action the metal would have if it were placed in the medullary cavity. Next they tried to see what action the metals would have if they were placed at the site of a fracture. They found that all the metals excited some degree of destruction which retarded growth of bone. This action also was essentially independent of location whether the metal was placed in the medullary cavity or directly on the bone surface. They tried to demonstrate that cellular reaction and calcification were inversely proportional to each other and that a metal which affected one did not affect the other.

In all these previous experiments, interpretations of results have depended upon macroscopic findings, tissue reactions, microscopic study which was but a magnification of the macroscopic detail, and some roentgenologic studies. Table I is arranged to show the wide variance of opinions resulting from these studies and the many inconsistencies thus far expressed as to the reactions of bone to metals.

Because of the wide difference of opinion resulting from former experiments and because of certain unpredictable clinical phenomena when metals are used in bone surgery, we assumed that there must be a variable factor yet undetermined. Our experiments therefore were undertaken to study the possibility that electrolysis might be the variable factor. In order to demonstrate this it was necessary first to study the reaction of normal bone to

TABLE I

CONTRADICTORY CONCLUSIONS OF VARIOUS AUTHORS ON THE EFFECTS OF METALS ON BONE

Metals Used	Metal Is Resorbed	Metal Is Not Resorbed	Indifferent Reaction	Partially Indifferent Reaction	Bone Growth Stimulated	Toxic Reaction on Bone	Bone Growth Inhibited	Bone Reaction Favorable
Aluminum	Duval,[5] Elsberg and Danborn[6]	Zierold,[21] Cretin[2]			Cretin[2]		Cretin[2]	
Silver		Metal oxidized	Lemerle[15]	Zierold,[21] Cretin[2]				
Copper		Rugh[19]	Cretin[2]		Cretin,[2] Zierold[21] (distant)	Cretin,[2] Zierold[21] (contact)		
Iron		Rugh[19]	Rolland,[18] Trout[20]			Leriche and Policard,[16] Cretin[2]	Zierold[21]	Lange[12]
Magnesium	All observers				Lambotte,[10,11] Cretin[2]		Zierold,[21] Cretin[2]	
Nickel	Zierold[21]	Rugh[19]	Potarca,[17] Cretin[2]			Zierold,[21] Cretin[2]	Zierold[21]	Hey-Groves[8]
Lead			Zierold,[21] Cretin[2]			Cretin[2]		
Zinc		Rugh[19]		Rolland[18]	LeGrande,[14] Cretin[2]		Zierold,[21] Cretin[2]	
Steel	Zierold[21]						Zierold[21]	
Stellite			Zierold[21]					Zierold[21]
Chrome-nickel			Jones and Lieberman[9]					Jones and Lieberman[9]

various basic metals, plated metals, or combination of metals (alloys) in a control series of dogs. Two screws made of similar metal were placed in the bone where they would be bathed by the same electrolyte (body fluids) and where no electrolytic action would be expected.

In two subsequent series of dogs, screws of these same metals were placed in duo in the bones in different combinations to find whether or not there would be an electrolytic reaction between them. As the experiments proceeded, we performed biochemical examinations of the tissues and tissue fluids, screws, liver, and in some instances kidneys, of the dogs as they were sacrificed. This was done because we observed that simple macroscopic, microscopic and roentgenologic observations were too variable and inconclusive to be of value. It is obvious that the proof of electrolysis of tissue depends upon evidence of electrolytic action according to the laws of the electromotive force of metals either in the solution about the individual screws or upon the interchange of metal ions between the screws. A criterion for proof of electric activity was that, in the presence of different metals in duo in the same bone, if ions of one metal should be carried to the other, according to the laws of the order of electromotive force of metals, it would be due to electrolysis. The body fluids in these experiments acted as the electrolyte and biochemical analyses were performed to demonstrate the presence

or absence in the electrolyte of one or the other of the metals in solution adjacent to, or adherent to, the other metal, ad seriatum, in accord with this law.

TABLE II

THE ORDER OF THE ELECTROMOTIVE FORCE OF METALS

Metal	Electromotive Force (E.M.F.)	Conductivity	Melting Point Degrees C.
Aluminum	1.7000	324,000	659
Zinc	0.7618	186,000	419
Chromium	0.5570	83,200	1,615
Iron	0.4410	63,000	1,535
Cadmium	0.4010	95,000	321
Cobalt	0.2780	10,300	1,478
Nickel	0.2310	144,200	1,452
Tin	0.1360	76,600	232
Lead	0.1220	50,400	327.4
Antimony	0.1000	27,100	630
Copper	0.3440	591,000	1,083
Silver	0.7978	681,200	960.5
Platinum	0.8630	91,200	1,773.5
Gold	1.3600	468,200	1,062

In all of these experiments, of which upwards of 50 were made, each radius of each dog was used. In the right foreleg the radius was fractured obliquely with an osteotome (to produce a condition of traumatized bone), and the fracture fixed with the two screws. The left radius was not fractured and two screws were placed in the bone similarly to those in the fractured right radius. The wounds were closed, the legs were fixed in plaster encasements, and the dogs maintained under especially favorable conditions for periods of four, six, and eight weeks.

At the time the dogs were sacrificed the macroscopic changes in the tissues were noted and photographed in color. Roentgenologic examinations of the legs were made, also biochemical studies of tissues, of liquid exudates adjacent to the screw holes, and of the screws themselves. Chemical examinations of the livers and kidneys were also made in many of the experiments. Finally sections of bone, at the screw holes, were removed for microscopic study.

In several of the experiments we found evidence of a migration of ions of one metal to another, which in each case was in accordance with the sequence of the electromotive force of metals. We found in a few instances an actual deposit of particles of one metal upon another. In one animal the transference of ions had involved two separate processes: a deposit of the copper ions from a brass screw onto the chromium of the chromium-plated screw and a deposit of chromium ions from this latter screw onto the zinc

of the brass screw, all in accord with the order of electromotive force of metals. In several experiments, we found chromium in the livers and kidneys of dogs in which we had used the chromium-plated screws, and copper in large excess in the livers and kidneys in which brass or copper screws had been used. In dogs into which any form of steel had been inserted, whether plain or plated or in alloy, there was an excess of iron in the liver. Two of the dogs that died 15 to 20 days after operation had had chromium-plated steel screws used and a large amount of chromium was found in their livers. It is possible these deaths were due to chromium poisoning, since there was no other cause found at the autopsy, which included a careful analysis of the stomach contents.

In determining the electrochemical character of a given combination of metals, the volume of each piece of metal, the distance between them and the difference in potential of the electrolyte must be taken into detailed consideration. Thus the body fluids of each individual, though essentially the same qualitatively, differ quantitatively.

When pure metals are used alone in the body, there is some direct chemical action by the body fluids. In the instance that only one metal is used, any ensuing structural changes are due solely to chemical influence, because, of course, no electrolysis can occur in the presence of only one metal. But if impure metals are used in the body, singly or with other metals, there is a greater tissue reaction due to electrolysis between the various ions.

Thus our experiments with plated metals showed the greatest reaction of all because it was from plated metals that we were able to demonstrate the transference of metallic ions from one plated screw to the substance of another screw of different metal in compliance with the law of E.M.F. The reason is that when plating becomes eroded or chipped the body fluids can come in contact with the two metals side by side and a battery is at once established. This is forcibly illustrated in our experiments with chromium-plated steel screws.

Proof of electrolytic activity in the tissues is the fact that we have been able to recover chromium from the zinc of a brass screw, copper from the chromium of a chromium-plated steel screw, copper from a silver-plated steel screw and zinc from the substance about a galvanized iron screw, *etc.*, which is shown in the detailed description of the experiments. In such cases, the very great reaction of soft tissue and destruction of bone at the screw site we believe to be due to the electrolytic action engendered by two metals rather than to the intolerance of the tissue and bone to a single meal. For instance, a galvanized iron screw is a mixture of iron and zinc, about which there is invariably a marked reaction, whereas there is little change in the tissues about either iron or zinc alone. This fact has confused many other observers since they could not explain the great variance in the changes that were discovered.

In the use of alloys of "rustless steel" which are made of metals more resistant to body fluids it is impossible to prove electrolytic action by the

transfer of the integral metals of one onto the metals of the other. Such alloys are actually different metals. But in direct accord with the law of electromotive force of metals, if there is a marked disturbance about an alloy due to one element being put into solution in the given electrolyte, the action is undoubtedly due to electrolysis and the effect is due to hysteresis. (Hysteresis is a molecular disintegration of metals in an alloy due to variations of local electrical resistance.)

We have accepted the findings of Zierold[21] and of Jones and Lieberman[9] in their reports of their experiments with the rustless steels, and believe with them that these new metals, for such these alloys are, are more resistant to body fluids and so cause less reaction. But we differ with them in their resulting recommendations because of the presence in all rustless steels of such a high percentage of iron, which is so subject to the action of body fluids and which sets up hysteresis and local electrolytic changes in the alloys. Our experiments with an alloy called vitalium, which contains no iron, showed complete resistance to body fluids and no changes whatever in either the surrounding tissues or in the bone. This metal was used in many combinations

TABLE III

CHEMICAL COMPOSITION OF VARIOUS ALLOYS USED IN BONE WORK

STELLITE	"NICKEL FREE" RUSTLESS STEEL	"HIGH NICKEL" RUSTLESS STEEL	"LOW NICKEL" RUSTLESS STEEL	VITALIUM	PROPOSED POSSIBLE ALLOY
Cobalt 58%	Chromium 15%	Chromium 8%	Chromium 18%	Cobalt 65%	Cobalt 65%
Chromium 35%	Manganese .4%	Nickel 22%	Nickel 9%	Chromium 30%	Vanadium 30%
Tungsten 4%	Silicon .3%	Copper 1%	Manganese .4%	Molybdenum 5%	Molybdenum 5%
Iron 3%	Sulphur .5%	Silicon 1.5%	Iron 70%	Manganese	Manganese
Carbon	Molybdenum .5%	Phosphorus	Carbon	Silicon	Silicon
	Carbon	Carbon			
	Phosphorus	Iron 65%			
	Iron 80%				
Contains iron, so increases electroactivity in alloy with added variance of E.M.F. because iron more subjected to action of electrolytic organic acids.	Chromium 15% 0.5570 E.M.F.	Chromium 8% 0.5570 E.M.F.	Chromium 18%	Chromium 0.5570 E.M.F.	
	Iron 0.4410 E.M.F. about 80%	Iron 65% 0.4410 E.M.F.	Iron 70%	Cobalt 0.2780 E.M.F.	
	Containing high per cent of iron	Nickel 22% 0.2310 E.M.F.	Nickel 9%	Molybdenum Conductivity	
		Conductivity Chromium 83,200 Iron 63,000 Nickel 144,000	Lower per cent of chromium and nickel less E.M.F.	Chromium 83,200	
		High electrolyte potential with variance conductivity	Smaller per cent of nickel less conductive potentiality	Cobalt 10,300 Melting Points Chromium 1615	
				Cobalt 1478	
				No iron	
				Least subject to action of electrolyte	

with other metals with uniform results as may be seen in the detailed report of our experiments.

These experiments show that the various chemical, tissue, and bone reactions are changes due to electrolysis, when two metals are used in couples in the same bone, and explain why there has been so much tissue disturbance locally and such variable results clinically.

In clinical orthopedic work electric couples are created by the use of plates, bands, wires, washers, lock nuts, *etc.*, in conjunction with screws, nails, or other appliances so that two different pieces of metal are used in the same bone. Here Ohm's law becomes effective; *i.e.*, that an electric current is directly proportionate to electric conductivity and inversely proportionate to resistance. Conversely, metals that are more nearly of the same electric conductivity offer less resistance to a current between them.

For example: The usual Lane plate furnished by instrument dealers is nickel-plated or chromium-plated steel, in which the iron is exposed at the holes into which screws of plain steel, chromium-plated steel, rustless steel, or galvanized iron are to be placed. What happens? Immediately an electrolytic action is started at the point of contact of each screw with the metal plate and the current is carried by the contacting plate to each other screw. There is then produced a progressive proliferative reaction in the tissues that is protective against the current. A necrosis and destruction of bone cells also takes place and an accumulation of discolored exudate, which contains elements of the metals in solution, forms about the metals and enters the circulation. In two weeks these screws, which were placed in the bone with forcible resistance, may be picked out with the fingers.

As we have shown, we have recovered chromium from the livers of dogs that had only small chromium-plated steel screws in each radius. The suggestion implied by this finding, which was incidental, seems very important because of the extreme toxicity of chromium. Looking back on our clinical experience, we are sure we have seen patients, in whom devices plated with chromium had been introduced, become much more ill than seemed warranted by the operation itself, which makes one wonder if chromium poisoning may not have been responsible. We feel that further investigation of this factor is important because of the wide and indiscriminate use of chromium in plating or alloy in so many appliances used in osteosynthesis.

Wire nails and galvanized nails are made of iron and zinc, and possibly some lead; copper nails and screws are usually copper with zinc or iron; steel screws commonly contain some zinc, while galvanized screws are usually made of iron plated with zinc. All of such metal devices as these have an electrolytic action whether used singly or in multiple, and if they are coupled together by like, or different material, the action is, in electrical terms, "stepped up."

Most of the nickel-plated and chromium-plated appliances sold today are not even electroplated, but instead are plated by chemical dipping. The outer coating of metal is easily chipped or rubbed off, or if the appliance is drilled

after plating, the two metals are exposed to direct contact at the screw holes with resulting electrolytic action. Such an assortment of materials courts the disaster we have so often seen clinically.

Electrolytic reaction to the different metals causes the collection of thin, brownish serous exudate about the site of the foreign metal *in vivo*, which, when chemically tested, contains the salts of the metals present in solution. The fluid collection is not due to infection, because this fluid, as well as the surrounding tissue, is microscopically bacteria free. As we have said, though, when metal ions from one screw are recoverable from another of different metal consistently with the law of E.M.F., it is due to electrolytic action and not solely to the chemical action of body fluids upon the metals themselves.

When screws have been placed too far apart in the bone to allow direct interchange of metal ions, there is a local electrolytic action in the screw itself between the metals of which it is made. This can be proved by a biochemical analysis of the tissues about the sites of these screws. To us this explains the interference with healing of fractures in which metal fixation of bone has led to necrosis and destruction of osseous tissue.

Finally, we believe that an alloy should be sought and developed that is entirely inert in the presence of, and unaffected by, biologic salts, to the extent that there will be no electrolytic action: and that it shall have the strength to resist such stress or strain as it may have to bear.

EXPERIMENTAL PROCEDURES

In dogs anesthetized with intravenous nembutal, two screws, consisting of the various metals, alloys, and combinations as described, were placed in the bone about 1½ cm. apart. The right radius was fractured in all the animals and the left radius was not, in order to check the reaction in each dog with and without trauma of the bone. Before the screws were placed, drill holes about two-thirds the size of the screw were made, in order to obviate the possibility of undue or variable pressure.

After the operations all wounds were closed with silk and the extremities fixed in a plaster encasement. The dogs were kept for observation in a well drained, shady corral, and fed separately with a selected, balanced diet.

EXPERIMENTAL RESULTS

CASE REPORTS

Dog 1.—Operation May 29, 1936. *Chromium-plated* screws used throughout. Died June 22, 1936. Autopsy showed no infection, although there was extreme loss of weight. Stomach analysis was negative except for chromate test which showed suggestion of chromium poisoning—more suspicious in subsequent experiments.

Roentgenologic Examination.—Necrosis of bone around all the screw holes. There was considerable tissue reaction about both operative sites. The fracture of the right radius was united. All the screws were loose one plus.

Dog 2.—Operation May 30, 1936. *Plain Steel* screws used throughout. Sacrificed July 20, 1936.

Roentgenologic Examination.—*Right:* There was nonunion of the fracture with destruction of bone and proliferation of callus.

Left: Destruction of bone about the screws with proliferation of new bone over screw heads. All screws were loose four plus.

Autopsy.—*Macroscopic: Right:* Nonunion of fracture with inflammatory soft red tissue over screw head and about the fracture site for 2 or 3 cms.

Left: There was much bony destruction with soft tissue necrosis for ½ to 1 cm. beyond screw heads. Other bone normal. Much excess bone for about ½ cm. around the screw.

Dog 3.—Operation May 29, 1936. *Vanadium Steel* screws used throughout. Sacrificed July 29, 1936.

Roentgenologic Examination.—Fracture united. Slight reaction in the shaft with destruction of bone at the screw heads and proliferation of new bone.

Autopsy.—*Macroscopic: Left:* The screws were tight in their sockets and both were covered with much fibrous tissue. Rim of bone was beginning to cover heads of screws. No discoloration of soft tissue near proximal screw. Excessive scar tissue near distal screw head.

Right: The screw head was completely covered by loose, adherent transparent fibrous tissue with faint brownish discoloration. Screw was loose. Fracture was united without excess callus. Slight necrosis over the head of screw.

Dog 4.—Operation June 3, 1936. *Silver Wire* used throughout. Sacrificed August 7, 1936.

Roentgenologic Examination.—*Right:* Fracture was united. Excessive proliferation of new soft bone.

Left: Silver pegs had worked out of place. Destruction of bone at the sites of both pegs.

Autopsy.—*Macroscopic: Right:* There was firm bony union of the fracture with an excess of firm callus. There was a slight accumulation of tan-colored fluid in a bursal sac immediately over the wire head. Excessive fibrous tissue was present for about 1 Mm. about the wire.

Left: Both wires had apparently worked out of the bone and become encysted. No necrosis at the holes. Holes were nearly filled with proliferation of new bone. Light brown discolored fluid about the wires. No infection.

Dog 5.—Operation June 3, 1936. *Silver-Plated Steel* screws used throughout. Sacrificed August 7, 1936.

Roentgenologic Examination.—Showed necrosis of bone at all screw sites with a marked proliferation of soft callus.

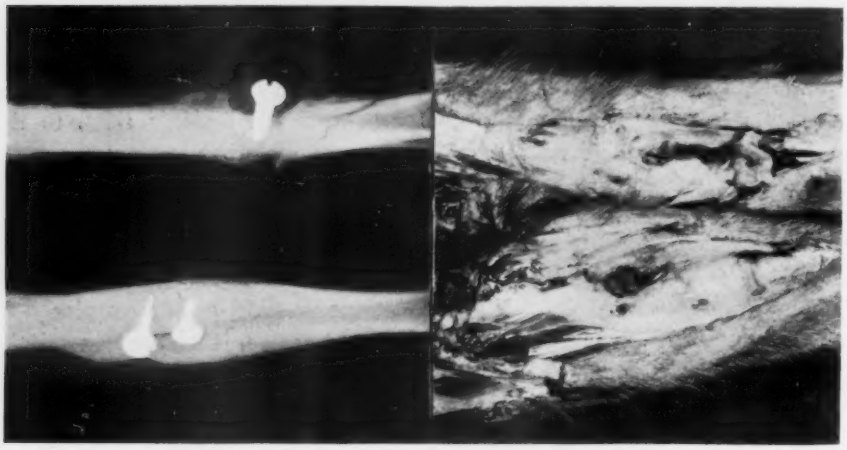

FIG. 1.—Dog 5: Silver-plated steel throughout. Roentgenogram showing marked necrosis of bone at all screw sites; note destruction about single screw.

FIG. 2.—Dog 5: Silver-plated steel throughout. Showing tissue reaction: Fracture ununited. Soft encapsulated seminecrotic tissue; tannish discolorations around heads of screws, all of which were very loose.

Autopsy.—*Macroscopic: Right:* Fracture was ununited. Screw was loose. The hole was covered with an excess of fibrous tissue for 2½ Mm. There was soft seminecrotic tissue lining an encapsulated area over the head of the screw. There was a moderate increase of soft callus.

Left: There was an excess of soft callus about the screws. Proximal screw was very loose. Distal screw was engaged. Bluish-tan discoloration over heads of screws which were encapsulated by fibrous tissue 2 Mm. thick.

Dog 6.—Operation June 3, 1936. *Copper Nails* used throughout. Sacrificed August 5, 1936.

Roentgenologic Examination.—*Right:* Union of fracture. Peg had worked out of bone. Necrosis with excessive proliferation.

Left: Necrosis with excessive proliferation. All pegs were loose four plus.

Autopsy.—*Macroscopic: Right:* There was fibrous union of the fracture with excessive fibrous tissue about the fragments. The copper nails had been displaced and were resting parallel to the bone. There was an excessive amount of fibrous tissue about the operative wound. About one nail there was a good deal of tissue necrosis with brownish fluid and tan discoloration. No pus.

Left: There was destructive necrosis near the holes where the copper nails were placed. There was marked necrosis also about the heads of the nails for 1½ cm. Beyond this was an area of marked proliferation of new bone. There was an excess of fibrous tissue almost completely covering the heads of the nails. This extended to 1 Mm. beyond the head of the proximal copper nail. The same was true of the distal nail, although the hole was about twice the size of the nail. There was a slight soft tissue necrosis about the heads of both nails.

Dog 7.—Operation June 10, 1936. *Copper* nail distal and *Steel* screw proximal on the left side. Reversed in the fractured right radius. Died July 16, 1936.

NOTE: In all the subsequent experiments with different combinations of metals in the same bone the arrangement of proximal and distal screws was reversed in the two legs to avoid any possible effect of gravity, or of contact of the solution of metals.

Roentgenologic Examination.—*Right:* Union of fracture. Necrosis of screw site and screw loose three plus.

Left: Slight change. Screws loose one plus.

Autopsy.—*Macroscopic: Right:* There was a sinus in the operative wound with evidence of a small abscess at the operative site and nails had worked out of the bone. Fracture was solidly healed in spite of infection. Tissue was necrotic around the wound.

Left: The copper nail was tight in the bone. Its surface was shiny. There was no apparent tissue necrosis in the region. The steel nail was not tight in the bone and it appeared tarnished. The soft tissue about the steel nail was discolored a dirty gray. Also this tissue showed evidence of liquid necrosis.

Dog 8.—Operation June 10, 1936. *Copper nail* proximal and *galvanized iron screw* distal on the left. Reversed on the right. Died June 22, 1936. No autopsy, cause of death unknown.

Roentgenologic Examination.—Much destruction of bone.

Autopsy.—*Macroscopic:* In both legs there was much tissue proliferation with excess of tan-colored fluid. No infection.

Dog 9.—Operation June 10, 1936. *Chromium-plated* screw proximal, and *copper nail* distal on right. Reversed on the left.

Roentgenologic Examination.—Showed marked bony necrosis at all screw sites, more marked with much more destruction at site of copper nails.

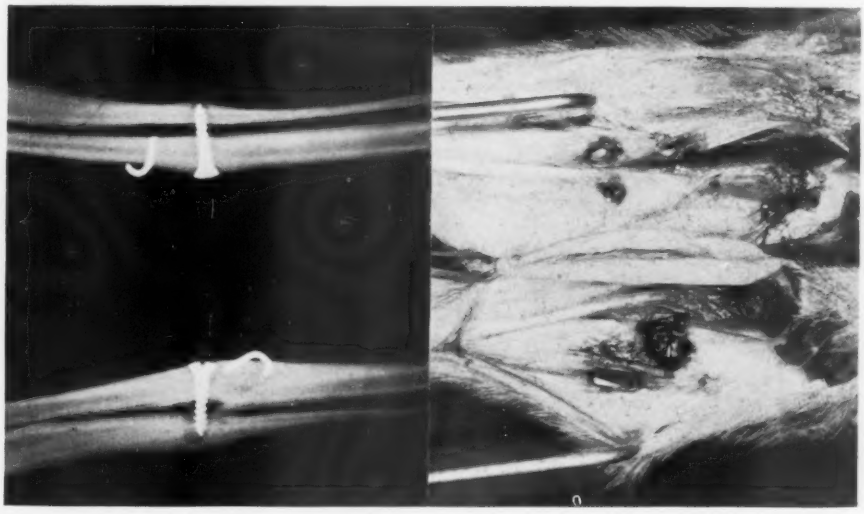

FIG. 3.—Dog 11: Silver and chromium-plated steel. Roentgenogram showing bone necrosis around the chromium but none is present adjacent to the silver.

FIG. 4.—Dog 11: Silver and chromium-plated steel. Showing tissue reaction: Marked reaction evident around the chromium plated steel screw which was loose. Very slight if any reaction present around the silver.

Autopsy.—*Macroscopic: Right:* Bony union of the fracture in good position. There was some destructive necrosis about the chromium-plated screw throughout its entire length. There was more destruction about the copper nail. There was apparently more new bone proliferation distal to the copper. Copper nail was loose. Necrosis over the head of it. Chromium-plated screw light. No excess callus or fibrous tissue.

Left: Decidedly more absorption of bone about the copper nail. Apparently no destruction about the chromium-plated screw. There was an excess of callus and fibrin about the copper nail. The screw was moderately loose and free in the hole. Slight amount of necrosis. The chromium-plated screw was tight. There was no excess of fibrous tissue and no discoloration.

Dog 10.—Operation June 13, 1936. *Plain steel screw* proximal, and *chromium-plated screw* distal on the right. Reversed on the left. Sacrificed June 21, 1936, because of compound fracture and gross infection. Not suitable for chemical study.

NOTE: At this stage of the experiments biochemical studies were begun of the tissues of the legs adjacent to the different screws, of the screws themselves, and of the livers and kidneys of the dogs as they were sacrificed.

Dog 11.—Operation June 13, 1936. *Chromium-plated screw* proximal, and *silver peg* distal on the left. Reversed on the right. Sacrificed September 1, 1936.

Roentgenologic Examination.—Showed marked necrosis about chromium-plated steel screws. None about the silver pegs.

Autopsy.—*Macroscopic: Right:* Around the distal chromium-plated screw there was soft reddish-tan fibrous tissue. Screw was loose two plus. Brown discoloration around the margin of the hole with

excess callus. About the proximal silver peg there was no discoloration, no necrosis, no looseness, no callus.

Left: The head of the proximal chromium-plated screw was completely covered flush with callus. There was very dark brown necrotic material over the screw head. Soft tissue around the bone was dark brown and necrotic. Screw was loose three plus. The distal silver peg was out of the hole in the bone and was encapsulated in soft fibrous tissue.

Biochemical Analysis: The soft tissues of the leg were positive for chromium about the chromium-plated screw on the left. The liver and both kidneys showed positive chromium tests.

NOTE: The discovery of chromium in the liver in this dog suggested the possibility that Dog 1 may also have died of chromium poisoning, particularly since the stomach contents showed a positive chromate test although strychnine was not present.

Histologic Examination.—Right proximal screw; silver: Moderate periosteal reaction with formation of moderate amount of chronic granulation tissue. No suppuration present. No evidence of bone regeneration about edges of screw hole. Moderate amount of bone degeneration present, however.

Right distal screw; chromium-steel: No periosteal reaction noted. No granulation tissue formation present. Bone undergoing marked degenerative changes.

Left proximal screw; chromium-steel: Marked periosteal reaction around edges of screw hole with thickening and increased vascularity. Large collections of plasma cells, lymphocytes and multinucleated cells. Marked production of granulation tissue. Some bone degeneration. No osteoplastic activity noted.

Left distal screw; silver: Marked granulation tissue formation with thickening and increased vascularity of periosteum. Some callus formation with moderate osteoplastic activity and with numerous

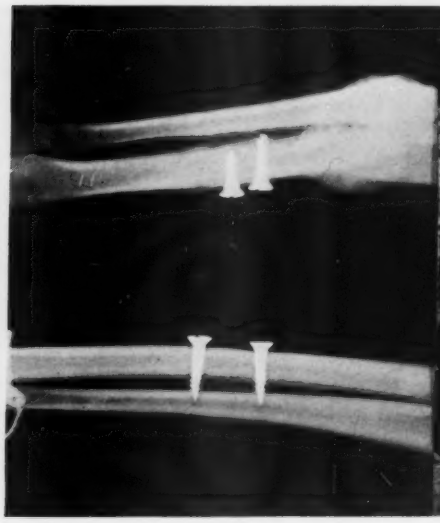

FIG. 5.—Dog 12: Silver-plated copper and steel. Roentgenogram showing moderate bone necrosis evident around all the screws.

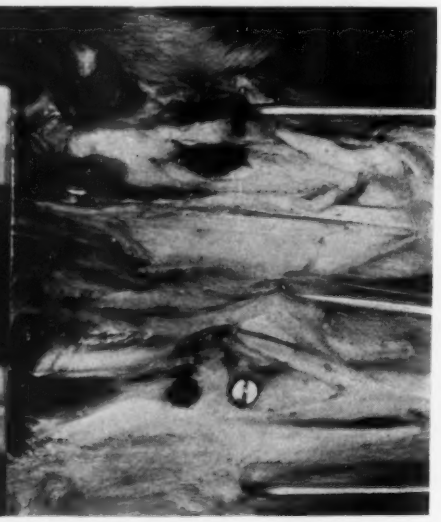

FIG. 6.—Dog 12: Silver-plated copper and steel. Showing tissue reaction: There is tan to black discoloration about the silver-plated copper screws. All screws are loose except the steel one in fractured radius.

osteoblasts scattered throughout granulation tissue. In one area there appears to be a chronic suppurative process.

Dog 12.—Operation June 13, 1936. *Plain Steel screw* proximal, and *silver-plated copper screw* distal on the left. Reversed on the right. Sacrificed September 1, 1936.

Roentgenologic Examination.—There was union of the fracture with moderate necrosis about all screws.

Autopsy.—*Macroscopic: Right:* In the bone about the proximal silver-plated copper screw there was brownish black discoloration and semifluid degeneration. Screw was loose one plus. Around the distal steel screw there was black discoloration. The screw was tight and there was no fluid.

Left: About the distal silver-plated copper screw there was much thin, pale tan fluid with similar tan discoloration of the tissue. Screw was very loose: three plus. There was liquefying necrosis. About the proximal steel screw there was black discoloration but no fluid. Screw was loose two plus.

Biochemical Analysis: There was an excess of copper in the liver and kidneys. There was erosion of the silver-plated copper screw in several places with traces of copper in solution in the adjacent tissues.

Histologic Examination.—Right proximal screw; Steel: Mild general reaction with small amount of granulation tissue formation. Mild bone necrosis. Regenerative changes slight.

Right distal screw; Silver-plated copper: Mild degree of granulation tissue formation. Mild degree bony degeneration. General reaction very slight.

Left proximal screw; Silver-plated copper: Moderate degree of bone necrosis. No evidence of granulation tissue formation or callus formation.

Left distal screw; steel: Marked granulation tissue formation. Callus formation with some bony regeneration present.

Dog 13.—Operation July 28, 1936. *Vitalium screw* proximal and *Vanadium Steel screw* distal on the right. Reversed on the left. Sacrificed August 7, 1936, because of severe screw worm infection of mouth.

Roentgenologic Examination.—Fracture united with solid bony union. Vanadium steel screws loose. Vitalium screws firmly in place. In the left leg there was no bony necrosis about the vitalium screw. About steel screws much bone necrosis and tissue reaction.

Autopsy.—*Macroscopic: Right:* Fractured ulna found to be solidly healed. Both vanadium steel screws were in place snugly. Vitalium screws were very tight and considerable force was required to remove them. There was very slight soft tissue reaction about the vanadium steel and none about the vitalium.

Left: Essentially the same findings as on the right.

Biochemical: Examination negative.

Histologic Examination.—Right proximal screw; Vitalium: Periosteum moderately thickened, but no granulations noted. Periosteum dense, relatively acellular with few blood vessels present. No bone necrosis noted, but there is a large wedge of what appears to be new bone present. General tissue reaction is of slight intensity.

Right distal screw; vanadium-steel: Moderate degree of periosteal thickening with a moderate granulation tissue formation about edges of screw hole. This tissue is undergoing extensive liquefaction necrosis. A few small foreign body giant cells are noted along junction of periosteum and granulation tissue. No bone necrosis is noted. General tissue reaction is one of moderate degree.

Left proximal screw; vanadium-steel: Mild periosteal reaction with a very small amount of granulation tissue present. Tissue reaction slight although there appears to be a slight amount of bone necrosis.

Left distal screw; vitalium: There appears to be little tissue reaction of any character, although a small amount of granulation tissue is observed. No bone necrosis.

Dog 14.—Operation August 21, 1936. *Galvanized iron screws* used throughout. Sacrificed November 2, 1936.

Roentgenologic Examination.—*Right:* Fracture ununited and angulated. Much necrosis and bony atrophy. All screws loose four plus.

Left: Necrosis at the screw sites. Screws loose four plus. No new bone growth.

Autopsy.—*Macroscopic: Right:* There was nonunion of the fracture in poor position. There was much fibrous tissue over the ends of the bone. About the proximal screw there was much destruction of bone. The distal screw was out of the bone in a cavity surrounded by amber fluid.

Left: There was very little proliferation of soft fibrous tissue with some pale tan discoloration. Both screws were loose four plus, with destruction of bone about the tips of the screws. Periosteum about the margin of the screw heads and between the screws was completely destroyed.

Biochemical Analysis: Zinc present in adjacent fluids. Liver was not tested for zinc.

Dog 15.—Operation August 21, 1936. *Vitalium screw* was placed proximally, and *galvanized iron* distally on left. Reversed in right leg. Died September 17, 1936. Cause of death unknown.

Roentgenologic Examination.—*Right:* Union of fracture. Destruction about proximal screw. No change about distal vitalium screw.

Left: No destruction near proximal screw. Destruction about galvanized screw.

Autopsy.—*Macroscopic: Right:* There was an hematoma in the leg about the proximal galvanized iron screw and the screw was loose two plus. The distal vitalium screw was tight.

Left: There was a small abscess over both screws in the field of the operation. The vitalium screw was covered with a transparent membrane and both screws were tight.

Biochemical Analysis: Negative.

Dog 16.—Operation July 18, 1936. *Chromium-plated steel screws* used throughout. Sacrificed September 8, 1936.

Roentgenologic Examination.—Showed moderate destruction of bone about heads of all screws. Bony union of fracture.

Autopsy.—*Macroscopic: Right:* The fracture was solidly healed and there was deep brown discoloration about both screws. The proximal screw was tight. The distal screw was covered with brown transparent areolar tissue.

Left: The distal screw had marked black discoloration of the tissue for a wide area. The screw was tight, although there was destruction of bone about the head of the screw from 3 or 4 Mm. The proximal screw was tight with destruction of bone for 1 Mm. about the head.

Biochemical Analysis: Much chromium in soft tissues adjacent to screw. Trace of chromium in the liver.

Histologic Examination.—Chromium plated steel screws throughout.

Right proximal: Moderate degree of periosteal thickening which has a shredded appearance. Small amount of granulation tissue present. Mild degree of bone necrosis.

Right distal: Mild degree periosteal thickening. No granulation tissue formation present. No bone necrosis—irritative changes slight in degree.

Left proximal: Periosteum mildly thickened with a moderate degree of granulation tissue formation which is undergoing organization. A few foreign body giant cells are scattered through the granulation tissue. General irritative changes are of moderate degree.

Left distal: Moderate degree of periosteal thickening with a moderate degree of granulation tissue formation. No bone necrosis noted. General tissue reaction is slight to moderate.

Dog 17.—Operation July 18, 1936. *Plain Steel screw* proximal and *brass screw* distal on the left. Reversed on right. Sacrificed September 9, 1936.

Roentgenologic Examination.—Union of fracture. Destruction of bone about all screws, more marked about the brass.

Autopsy.—*Macroscopic: Right:* The distal steel screw was tight with some fibrous tissue over the

head. The proximal brass screw was loose two plus with a slight amount of boggy tissue over the screw head.

Left: Both screws were very loose four plus. There was much new bone about both screws with erosion about the heads and much brownish discoloration over the proximal steel screw.

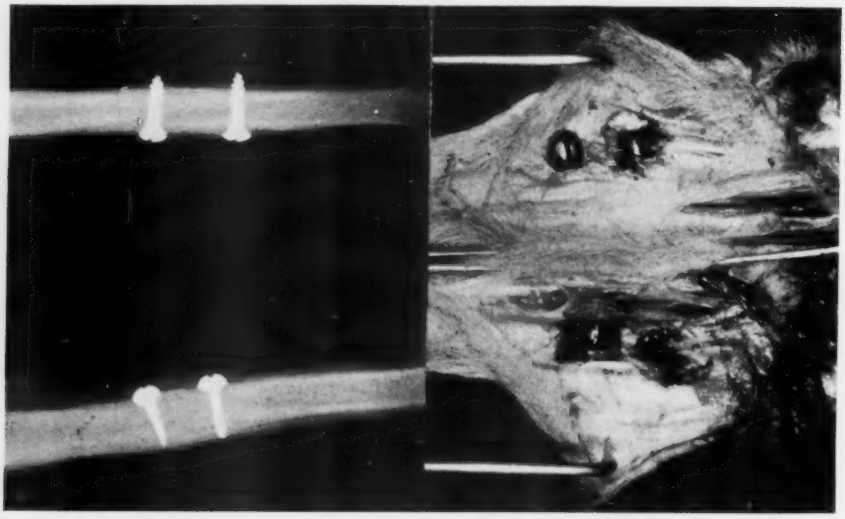

FIG. 7.—Dog 16: Chromium-plated steel. Roengtenogram showing moderate necrosis of bone about all the screws.

FIG. 8.—Dog 16: Chromium-plated steel. Showing tissue reaction: There is marked local reaction around all screws with excess proliferation and dark discoloration. The screws are tight.

Biochemical Analysis: Iron and copper present in the soft tissues adjacent to the screws. Greater quantity present on the left side because the screws were closer together. Much iron found in the liver.

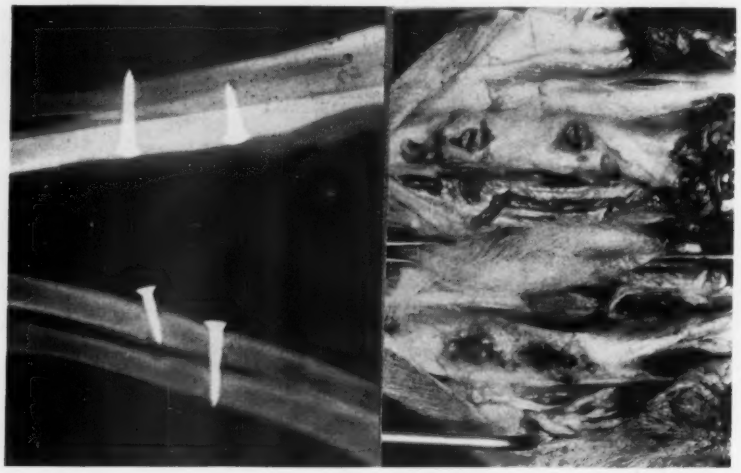

FIG. 9.—Dog 17: Steel and brass. Roentgenogram showing destruction of bone about all the screws with marked necrosis around the brass.

FIG. 10.—Dog 17: Steel and brass. Showing tissue reaction: There is much new soft bone with marked fibrous proliferation. All the screws are very loose except the steel one in the fractured radius.

Histologic Examination.—Right proximal screw; brass: Moderate amount granulation tissue formation with marked periosteal thickening. No bone necrosis. Some new cartilage present.

Right distal screw; Steel: Marked thickening and fibrosis of periosteum with moderate osteoblastic

activity. Moderate degree of granulation tissue formation. No bone necrosis. Irritative changes not marked.

Left proximal screw; steel: Marked periosteal thickening and fibrosis with a very slight amount of granulation tissue formation present. No bone necrosis. Numerous osteoblasts present.

Left distal screw; brass: Marked callus formation with thickening of periosteum. No granulation tissue formation noted or has become organized into callus. New cartilage formation noted. Few irritative changes present.

Dog 18.—Operation July 18, 1936. *Brass screw* proximal and *galvanized iron* screw distal on the left. Reversed on the right. Sacrificed September 9, 1936.

Roentgenologic Examination.—Firm union of fracture. All screws loose except proximal brass in left. Much destructive necrosis of bone at all screw sites.

Autopsy.—*Macroscopic: Right:* The distal brass screw was loose two plus. There was no necrotic material. No fluid and moderate proliferation of new bone. The proximal galvanized iron screw was loose four-plus with a proliferation of new bone about the margin of the screw head.

Left: The proximal brass screw was firmly fixed in the bone. The distal galvanized iron screw was loose four-plus. There was much new bone about both screws with more about the proximal. There was brownish discoloration of the seminecrotic material around the distal screw.

Biochemical Analysis: Liver showed heavy trace of copper. Trace of copper and zinc in tissue adjacent to the screws.

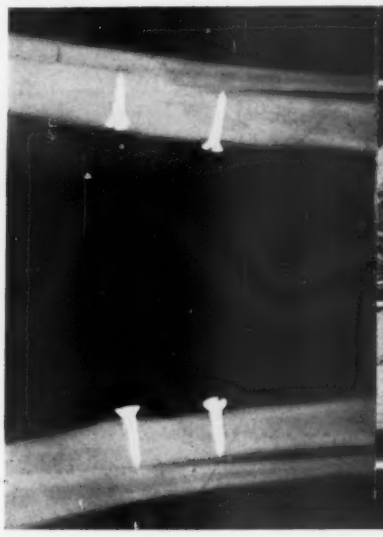

FIG. 11.—Dog 18: Galvanized iron and brass. Roentgenogram showing marked bone necrosis around all screws.

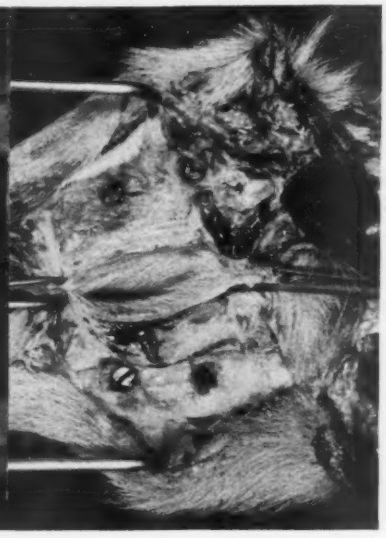

FIG. 12.—Dog 18: Galvanized iron and brass. Showing tissue reaction: There is present brown discoloration with proliferation of soft, fibrous tissue. All screws loose.

Histologic Examination.—Right distal screw; brass: Moderate degree of periosteal thickening around screw hole. No granulations present. No irritative reaction. No callus or bony regeneration. Process appears generally indolent with some necrosis in tissue lining the screw hole.

Left distal screw; galvanized iron: Mild degree of periosteal thickening around edges of screw hole with a marked degree of granulation tissue formation. Numerous large cells (osteoblasts?) scattered through granulation tissue which contains numerous inflammatory cells. Appearance is that of a marked tissue reaction of irritative character.

Dog 19.—Operation July 21, 1936. *Plain Steel screw* proximal and *vitalium screw* distal on the left. Reversed on the right. Sacrificed September 9, 1936.

Roentgenologic Examination.—Union of fracture. Distal steel screw on the right loose four plus with erosion about the head. Vitalium caused no reaction. On the left the vitalium screw was slightly loose, and the steel screw was loose three plus. There was a ridge of new bone 2 Mm. high on the medial side of the steel screw.

Autopsy.—*Macroscopic: Right:* The proximal vitalium screw was about 50 per cent covered with normal new bone. The screw threads were intact and the screw could be unscrewed easily. There was no discoloration and no abnormal bone. The distal steel screw was loose three plus. There was much excessive new bone around the margin of the head with a mass of encapsulated brownish soft tissue over the head.

Left: The proximal steel screw was loose three plus with a small amount of soft brown discoloration of the adjacent tissue. The distal vitalium screw had worked loose. On the medial side of the hole was a ridge of new bone about 2 Mm. high (? torn periosteum). There was no discoloration.

Biochemical Analysis: Vitalium screws showed no trace of iron about them. About steel screw was

proliferation of iron into soft tissue. Excessive amount of iron in the liver. No trace of chromium or cobalt in the liver.

Histologic Examination.—Right proximal screw; steel: Little tissue reaction present. Periosteum

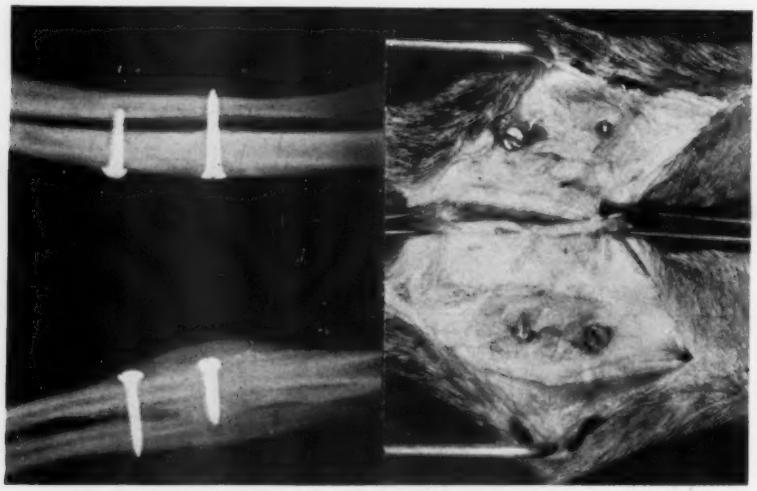

FIG. 13.—Dog 19: Vitalium and steel. Roentgenogram showing no bone changes about vitalium and erosion of bone around the steel screws.

FIG. 14.—Dog 19: Vitalium and steel. Showing tissue reaction: No changes about vitalium; one vitalium screw is one plus loose; marked reaction about all steel screws, which are very loose.

not thickened, dense and relatively acellular. Small amount of granulations present which appear partially necrotic.

Right distal screw; vitalium: Mild periosteal thickening, small amount of granulation tissue present. Marked regeneration of bone, probably along fracture site.

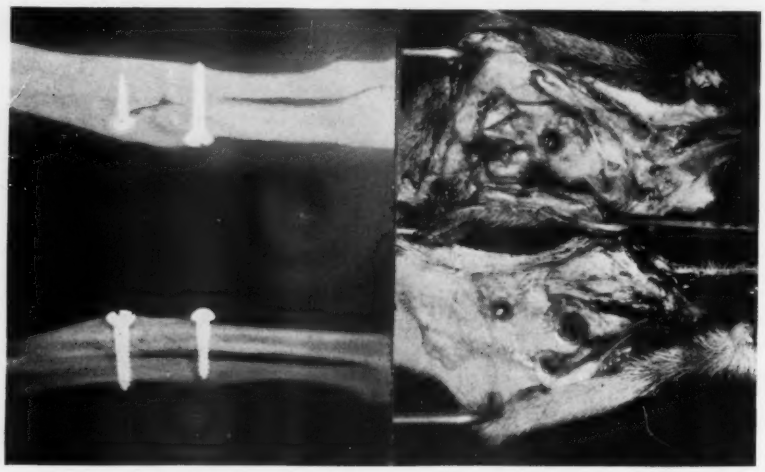

FIG. 15.—Dog 20: Vitalium and brass. Roentgenogram showing no change around vitalium screws; marked bone destruction about brass screws.

FIG. 16.—Dog 20: Vitalium and brass. Showing tissue reaction: No reaction adjacent to vitalium; tissue destruction with proliferation about both brass screws, which are four plus loose.

Left proximal screw; vitalium: No periosteal thickening noted. Periosteum covered with a moderately thick layer of granulation tissue which is undergoing early organization. No foreign body giant cells noted. General tissue reaction is not marked.

Left distal screw; steel: Moderate periosteal thickening with fibrocystic proliferation and formation

of a few foreign body giant cells near periosteal attachment to bone. Lining the screw hole is a thick layer of granulation tissue undergoing organization. General tissue reaction is marked.

Dog 20.—Operation July 21, 1936. *Brass screw* proximal and *vitalium screw* distal on the left. Reversed on the right. Sacrificed September 9, 1936.

Roentgenologic Examination.—Fracture solidly united. No bony changes about either vitalium screws and they were tightly in place. There was necrosis of bone about both brass screws which were loose.

Autopsy.—*Macroscopic: Right:* Over the distal brass screw there was a large mass of reddish brown encapsulated tissue. All the neighboring soft tissue was boggy. The proximal vitalium crew was loose four plus. There were no threads in the hole and the screw had apparently been placed between the fragments of the fracture. There was no bony proliferation about the head.

Left: The distal vitalium screw was very tight. There was no discoloration and no destruction of the bone. The proximal brass screw was loose four plus, with much necrotic material around the screw. There was little proliferation of new bone.

Biochemical Analysis: Copper in tissue adjacent to screws in right foreleg. Trace of copper in the liver.

Histologic Examination.—Right proximal screw; vitalium: Mild periosteal thickening about edges of screw hole, but no granulations noted. Process here appears relatively indolent with what appears

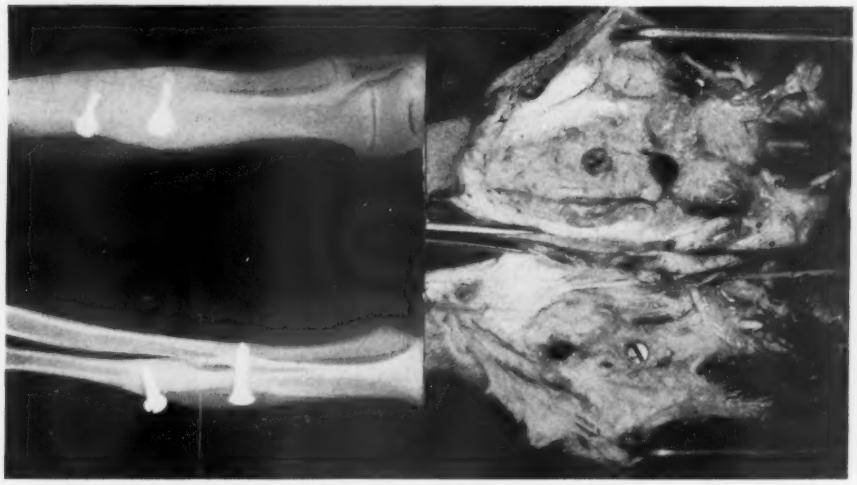

FIG. 17.—Dog 21: Galvanized iron and brass. Roentgenogram showing much destruction of bone about all screws.

FIG. 18.—Dog 21: Galvanized iron and brass. Showing tissue reaction: Soft brown granular tissue about galvanized-iron screw and soft fibrous tissue brass screws; all screws are loose except one of the brass ones.

to be a mild degree of necrosis taking place. The bony elements show no necrosis and deeper there is a wide, dense callus in process of formation.

Right distal screw; brass: Periosteum somewhat thickened, but is split up and shredded in appearance from exudative material. There appears to have been a marked granulation tissue formation but this is undergoing advanced liquefaction necrosis, and is not definitely recognizable as granulation tissue. There is a slight degree of bone degeneration just beneath periosteum. No new bone formation noted.

Left proximal screw; brass: Marked thickening and fibrosis of periosteum about edges of screw hole but only a very slight granulation tissue formation. No bone necrosis noted. No bone regeneration.

Left distal screw; vitalium: General tissue reaction very slight. Periosteum not thickened, but heavily infiltrated with cells similar to plasma cells and lymphocytes. A very slight granulation tissue formation is present. In one area at edges of screw hole there is a small area of bony necrosis while in another area there is a definite but slight degree of new bone formation.

Dog 21.—Operation July 21, 1936. *Brass screw* proximal and *galvanized iron screw* distal on left. Reversed on right. Sacrificed September 8, 1936.

Roentgenologic Examination.—Union at fracture site. All screws loose with much destruction of bone.

Autopsy.—*Macroscopic: Right:* Fracture solidly united. The proximal galvanized iron screw was surrounded by boggy tan granulation tissue on the proximal side. The screw was only slightly loose. The distal brass screw was tightly engaged with much slightly discolored new bone growth and dense fibrous tissue covering the screw.

Left: The proximal brass screw was loose two plus. There was some discoloration and erosion of the soft tissue about the screw but no fluid. The distal galvanized iron screw was loose three plus with

a small amount of free fluid about it. There was destruction of the bone without metallic discoloration and considerable boggy fibrous tissue about the screw head.

Biochemical Analysis: Much iron in soft tissue adjacent to screws. Trace of copper deposited on galvanized iron screw.

Histologic Examination.—Right distal screw; brass: Marked periosteal reaction with moderate thickening. Moderate degree of granulation tissue formation with almost complete organization. Moderately advanced callus formation deep in bone along probable fracture line with new bone formation taking place. General tissue reaction of moderate degree of intensity.

Right proximal screw; galvanized iron: Mild periosteal reaction with mild degree of thickening. Moderate degree of granulation tissue formation with a slight organization beginning to take place. Numerous degenerating bony fragments scattered through granulation tissue. Some evidence of bone regeneration present but this is not marked. General tissue reaction is of moderate degree.

NOTE: In Dogs *21* and *22* screws happened to be placed in closer approximation than in the other experiments.

Dog 22.—Operation July 28, 1936. *Brass screw* proximal and *chromium-plated screw* distal in left. Reversed on right side. Sacrificed August 17, 1936.

Roentgenologic Examination.—Union of fracture. All screws were loose and showed necrosis with excessive proliferation of new bone at all screw sites.

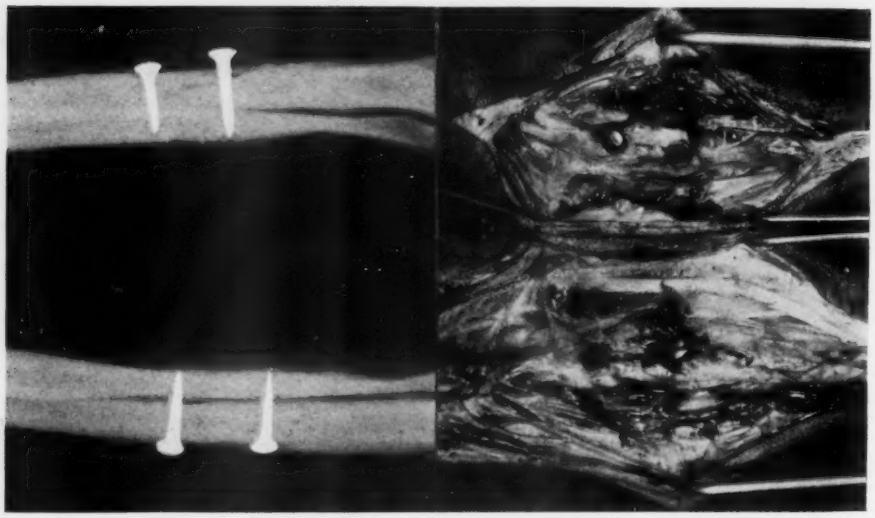

FIG. 19.—Dog *22*: Chromium-plated steel and brass. Roentgenogram showing proliferation of new soft bone with necrosis at all screw sites.

FIG. 20.—Dog *22*: Chromium-plated steel and brass. Showing tissue reaction: Black stain about the chromium-plated screws. Slight reaction about all screws, which are very loose.

Autopsy.—*Macroscopic: Right:* The periosteum near the fracture was bulging with an accumulation of brownish serous exudate. The fracture was united with excessive proliferation of new bone. The brass screw was loose and free to rotate. The chromium-plated screw had new bone about it but no destruction of periosteum. The screw was loose and there was a small area of erosion at the margin where it touched the bone. The edges of the hole were black.

Left: The brass screw was tightly engaged. The tissues seemed healthy except for an area about ½ cm. around the chromium-plated screw where there was a low-grade tissue necrosis with pinkish-gray secretion. There was a proliferation of fibrous tissue about the brass screw which seemed normal. About the chromium-plated screw there was no new bone growth and there was degenerative necrosis in the screw hole.

Biochemical Analysis: Copper was deposited on chromium-plated screws and chromium on the zinc of the brass screws in both legs. (NOTE: According to the Table of E.M.F. copper should be deposited on to chromium, chromium on to zinc.) Trace of chromium in the liver.

Histologic Examination.—Right proximal screw; chromium-plated steel: Moderate periosteal thickening with moderate degree of granulation tissue formation. Moderate number foreign body giant cells scattered through periosteum and granulation tissue. Marked bone regenerative changes present. General tissue reaction changes are marked.

Right distal screw; brass: Moderate periosteal thickening with moderate degree of granulation tissue formation. Moderate number foreign body giant cells scattered through periosteum and granulation tissue. Marked bone regenerative changes present. General tissue reaction changes are marked.

Left proximal screw; brass: Marked periosteal thickening with small amount of granulation tissue formation. Numerous foreign body giant cells in periosteum. Mild degree of bone degeneration present near margin of screw hole. Irritative tissue reaction moderate.

Left distal screw; chromium-plated steel: Marked degree of periosteal thickening with moderate

degree of granulation tissue formation. Moderate degree of new bone formation along line of fracture with marked callus formation. General tissue reaction moderate to marked.

Dog 23. Operation July 23, 1936. *Steel screw* proximal and *silver-plated copper screw* distal on left. Reversed on right. Sacrificed September 3, 1936.

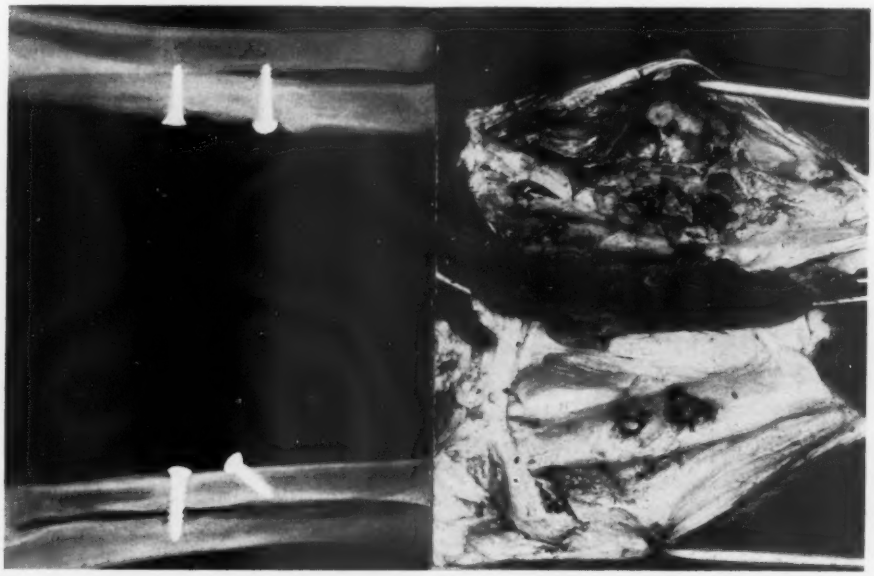

FIG. 21.—Dog 23: Silver-plated copper and steel. Roentgenogram showing proliferation of new bone at fracture site. Necrosis at all screw sites.

FIG. 22.—Dog 23: Silver-plated copper and brass. Showing tissue reaction: Gray exudation about the copper screws with brown discoloration about the brass screws. All screws loose.

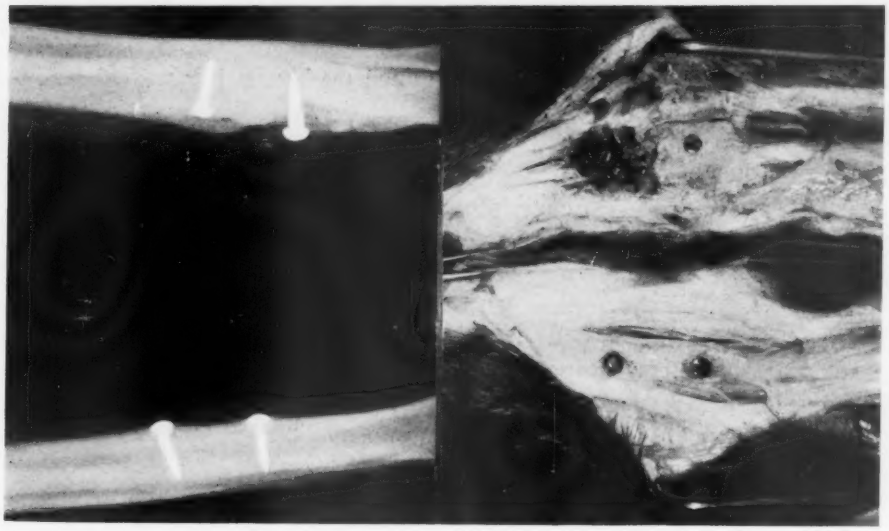

FIG. 23.—Dog 24: Vitalium throughout. Roentgenogram showing no change around screws except one in fracture site, about which there is new bone growth.

FIG. 24.—Dog 24: Vitalium throughout. Showing tissue reaction: Granulation around the one screw in fracture site; none about the remaining screws, all of which were very tight.

Roentgenologic Examination.—Union of fracture. All screws loose with slight necrosis and much new bone.

Autopsy.—*Macroscopic: Right:* There was a small amount of grayish secretion on the proximal

side of the distal steel screw. The head of the screw was completely covered with new bone. The silver-plated copper screw was loose two plus. There was a small amount of callus and some brownish fluid about the edges of the screw.

Left: There was an excess of brown tissue about the steel screw and the screw was loose two plus. The silver-plated copper screw was tightly engaged. Both screw heads were encapsulated and there was a small amount of tannish tissue about the head of each. There was some formation of new bone about the steel screw but the screw itself was loose in the hole.

Biochemical Analysis: Trace of copper about the steel on the left. Trace of copper in the liver.

Histologic Examination.—Right proximal screw; silver-plated copper: Slight to moderate degree of periosteal thickening. Granulation tissue formation slight to moderate with low grade inflammatory reaction. Numerous eosinophiles scattered throughout granulation tissue. General tissue reaction appears to be of moderate degree.

Right distal screw; steel: Marked thickening of periosteum which appears dense and fibrous. Moderate granulation tissue formation with a low grade chronic inflammatory process. Some hemorrhage and fibrinous exudation in deeper tissues. General irritative tissue reaction may be classed as moderate.

Left proximal screw; steel: Periosteum shows a slight to moderate degree of thickening with only a small amount of granulation present. No giant cells observed. General tissue reaction appears to be slight.

Dog 24.—Operation July 23, 1936. *Vitalium screws* used throughout. Sacrificed September 8, 1936.

Roentgenologic Examination.—There was union at fracture site. No reaction about screws except one in the fracture site, which showed proliferation of new bone. No inhibition of bone growth in fracture site.

Autopsy.—*Macroscopic: Right:* There was no infection, no discoloration, and the screws were bright. The proximal screw was embedded under 2 or 3 Mm. of dense healthy new bone. No fibrous tissue, no fluid. The distal screw was in the fracture site and was loose four plus. Serosanguineous fluid had collected in an area about 1/3 by 1½ cm.

Left: Both screws were tight and were covered with a thin normal transparent fascia. No discoloration. No destruction of the tissue.

Biochemical Analysis: No evidence of metal in soft tissue of the leg, liver or kidney.

Histologic Examination.—Vitalium throughout.

Right distal: Marked periosteal thickening with moderate to marked degree of granulation tissue formation which is undergoing organization. Marked bone regeneration along line of fracture. General tissue reaction is marked.

Right proximal: Moderate periosteal thickening. Very slight degree of granulation tissue formation. General tissue reaction slight.

CONCLUSIONS

(1) That it is impossible from macroscopic, microscopic and roentgenologic studies alone, to differentiate accurately the different reactions of soft tissue and bone to metals.

(2) That such reactions can be explained readily by biochemical study, based upon the behavior of metals ad seriatim in accordance with the laws of the electromotive force of metals.

(3) That electrical force is generated when different metals are placed in the tissues, by the creation of a battery, and the amount of current is proportionate to the difference in potential of metals at the two poles, the degree in which they are acted upon by the given electrolyte, and the distance between the poles.

(4) That pure metals alone are inert. Any action which takes place about them is a chemical reaction to body acids and has no electrolytic significance. Example—a battery containing two zinc plates only, cannot be "charged" electrically though the zinc may be taken into solution by the sulphuric acid.

(5) That those metals most widely separated in the E.M.F. series create the greatest potentials, although only one of the pair may be acted upon chemically by the electrolyte.

(6) That when different metals are connected or coupled directly, there is a galvanic action between the poles which is increased in direct ratio with the resistance of the couple, and the rate of corrosion on the more electropositive metal is accelerated. This is exactly analogous to a "short" in an ordinary wet cell.

(7) That any single metallic appliance made of galvanized iron, plated steel, or of metallic alloy containing a metal subject to the action of body fluids will cause some galvanic action.

(8) That it is this electrolytic action which causes the formation of irritating metallic salt solutions in the local fluids. The reaction against it leads to the excessive proliferation of cellular and fibrous tissue which is protective, and the inhibition of growth of bone which is destructive.

(9) That no different metals should be placed in a given patient and only pure metals, least subject to the action of the body fluids, should be used as far as possible. This explains why pure silver wire has been fairly well tolerated. However, when rigidity and tensile strength are necessary as in plates, screws, and nails, pure metals are too soft and alloys of the more resistant metals have to be used.

(10) That in the selection of alloys to be used the relative position of metals selected in the E.M.F. series is not essential as the properties of the alloy may be different from any of its components.

(11) That in our opinion such alloys should not contain iron because iron is so subject to the action of the physiologic body salts. In all of our experiments those metals which contained iron showed large amounts of iron in the tissues adjacent and an excess of iron in the livers.

(12) The alloy of least reaction that we have found is one called vitalium, which contains no iron and consists of cobalt, chromium and tungsten. There was no tissue reaction or bone change at the site of any vitalium screw though it was checked in many experiments with several different combinations of metals.

(13) That any appliance that is plated with chromium is dangerous because chromium salts, which are extremely poisonous, are promptly liberated and readily concentrated in the liver. Once the plating is chipped or broken, a battery is immediately created. For this reason, unless chromium in an alloy can be shown to have had its potential completely lost in the new potential of the alloy in its changed position in the E.M.F. series it might have the same effect as when used in plating.

(14) That for this reason we suggest an alloy for bone work that is similar to vitalium in which vanadium may be substituted for the chromium, if such an alloy should confer the same degree of corrosion resistance and not be too brittle. However, following our experiments with the alloy vitalium, the constituents of its components seem to have lost their potential, as there was no trace of chromium liberated in any instance.

We wish to express our appreciation to Drs. Milton Davis and D. A. Todd of the Nix Hospital Laboratories for their assistance in the roentgenologic studies and microscopic sections, to the San Antonio Analytical Laboratory for the biochemical study, to the Abbot Laboratories for the supply of nembutal for use in intravenous anesthesia, to the Austenal Laboratory of New York for making the screws of vitalium, and to the National Manufacturing Company of Sterling, Ill., for their kindness in supplying metal screws of all types.

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INTERNAL FIXATION IN FRACTURES OF THE NECK OF THE FEMUR

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IN ANY discussion of fractures of the neck of the femur, differentiation must be made between those which occur in the central or narrow portion of the neck (so-called intracapsular fractures) and those which occur in the region of the trochanter. Since the time of Sir Astley Cooper, differentiation has been made as to location, and at that time it was presumed that fractures within the capsule did not unite by bony union, but that those external to the capsule united rapidly by bony union, as excellent functional results were secured. Regardless of accurate knowledge of over a hundred years' duration of the fundamental principles involved, there is still misunderstanding about fractures in this region. Within the past year I have seen fractures through the neck and trochanter exhibited before a well known national association on two occasions, without differentiation, and within the past 10 years on more than one occasion before this association. Differentiation must also be made between complete central fractures of the neck of the femur and impacted central fractures, as the latter will unite without difficulty, and a good functional result will be obtained if the impaction is not disturbed.

In 1932, I[1] demonstrated that trochanteric fractures were of more frequent occurrence than complete central fractures and that impacted central fractures were not infrequent, after reviewing the records of 688 fractures of this region of which 495 were fresh fractures which may be enumerated as follows:

		Over 70 Yrs. of Age		
Trochanteric	229	108	average age	78.1%
Complete central	214	78	average age	74.6%
Impacted central	52	19	average age	79.5%

Thus, impacted fractures of the neck of the femur and intertrochanteric fractures constitute more than half of the fractures presented above (56.7 per cent), in which union may be expected in practically 100 per cent, if only approximate anatomic apposition is maintained. Only in central fractures of the neck of the femur (43.3 per cent) is there any doubt as to securing osseous union. The average age of trochanteric fractures as noted above is higher, and of even more frequent occurrence, in elderly individuals (70 years or older) than central or intracapsular fractures.

Therefore, especial emphasis must be placed upon differentiating between the location and characteristics of (1) complete, central or intracapsular, (2) impacted central fractures of the neck of the femur, and (3) fractures in

the trochanteric region, in any discussion of this most important subject. Regardless of our modern methods of diagnosis, the older surgeons who classified these fractures as intra- and extracapsular, had a better understanding of fractures of this region than is often manifested today.

As stated above, complete, central fractures of the neck of the femur constitute only 43 per cent of fractures in and about the neck and trochanters. This in no way disparages its importance or frequency of occurrence as fractures of this region are quite numerous. Prior to 1904, it was considered that central or intracapsular fractures did not unite by osseous union. At this time, Royal Whitman described the abduction method of reduction with fixation by a plaster of paris spica and demonstrated that osseous union could be secured. This procedure was soon adopted by the orthopedic surgeons of America and it was thought that the problem had been solved as a very high percentage of excellent functional results were reported. This was undoubtedly due to the fact that many impacted and trochanteric fractures were included in the analysis of cases, as Whitman himself did not clearly differentiate. However, as time elapsed and discrimination was made as to location and characteristics, a committee of the A.O.A., in 1929, made a very careful survey, excluding all except complete central or intracapsular fractures, and found that osseous union was secured in 50.4 per cent of 201 fractures from various well known clinics of America.

Obviously, the Whitman method was the first and possibly the greatest advance that has ever been made in the treatment of this fracture, but still left much to be desired, as long fixation and confinement were required before osseous union could be determined, often with permanent damage to the hip and knee or accompanied by serious constitutional complications.

The frequent occurrence of nonunion in complete central or intracapsular fractures is due to the physiologic status of the region, which has been described in detail in former contributions. The more important factors may be briefly mentioned, as: Impairment of circulation to the proximal fragment, osteoporosis, inaccurate reduction, shearing action of weight bearing, the deleterious action of the joint fluid on callus, and deficient blood clot.

In any treatment of fracture of the neck of the femur, accurate reduction is essential, but not until recently could this be determined by the roentgenogram, as only the anteroposterior view was possible. The lateral view is now obtained by the curved cassette and other methods, so that more exact relations can be determined. This development has undoubtedly been a great advance in the treatment of fractures of this region; however, I have observed, after taking both views, that reduction is accomplished by the Whitman method in practically every instance, so that I do not believe that this factor alone accounts for the many failures after this treatment.

Internal fixation by nails, screws, and pegs of this fracture is by no means new, as the first report was made by von Langenbeck[2] of one case treated by this method in 1850. Successful results were reported by Nicolaysen[3] and others prior to the advent of the roentgen ray. E. D. Martin and A. C.

King,[4] in 1922, and Thomas,[5] in 1921, report excellent results, using wood screws as a means of internal fixation. At this time internal fixation was not uniformly successful either because of inaccurate reductions or because of lack of dexterity on the part of the surgeon in introducing the means of fixation. In consequence, this method fell into disuse with the exception of a small number of surgeons who were probably more expert in properly placing internal fixation, notably E. Denegre Martin of this Association. Recently his end-results were reviewed by Dr. Brewster,[6] of New Orleans, who added 21 cases of his own treated by this method; he reports 89.68 per cent successful results in a series of 53 cases. The more recent literature abounds with various ingenious methods of obtaining internal fixation.

Smith-Petersen,[11] in 1925, introduced a three flanged nail which he placed through the neck of the femur by means of a wide exposure of the hip joint, and to him is undoubtedly due the credit for reviving interest in internal fixation which is rapidly becoming the method of choice. This nail has the advantage of securing fixation and preventing rotation. Many who have adopted this procedure believe that the operation is too formidable in the aged, and are applying the nail by inserting it through an incision over the trochanter, so-called "blind nailing," as it had been employed by other surgeons prior to Smith-Petersen. Local anesthesia may be successfully employed if indicated or desired. It has been proved that just as accurate fixation can be accomplished and maintained in this manner, and without any appreciable surgical shock. Since this method has become popular, other means of internal fixation have been employed, as: Multiple spikes (Gaenslen[7]), two pins (Knowles[8]), two drills (Rowlette, Haslem, Siegert, Morris, Key[9]), three pins threaded in outer half with a small nut (Moore[10]). All of these have two or more pegs which traverse the neck and head so as to prevent rotation of the head. In some, the head and neck of the femur become a veritable pin cushion. Excellent results have been reported by all of these methods; the problem is apparently a question of efficient internal fixation. The amount of foreign material, if pins or spikes are used, is approximately the same as the Smith-Petersen[11] nail, but has the advantage of being much less expensive.

No one has been a stronger advocate of the Whitman procedure than I, but after seeing the results of Smith-Petersen and others, I have come to the conclusion that internal fixation not only gives a higher percentage of excellent functional results and firm osseous union, but also materially decreases the time in which union is secured, so that weight bearing and walking without support is obtained in from four to six months as compared to six to 12 months by the Whitman method. Also function in the knee and hip is materially conserved, and permanently restricted motion is exceedingly rare. The mortality has been materially decreased by the less extensive means of immobilization and the shorter time of confinement to bed.

My colleagues and I have employed the three-flanged nail in 35 cases of complete, central, or intracapsular fractures of the neck of the femur. In 11, the procedure as described by Smith-Petersen was employed, which consisted

in a complete exposure of the hip joint and trochanter; in 24, the nail was inserted through a lateral incision with "blind nailing." The Smith-Petersen technic has been entirely discarded, as so-called "blind nailing" is a less extensive surgical procedure.

Operative Technic.—The present technic of insertion may be described as follows: Reduction is uniformly accomplished by the well known Whitman abduction method, so that, usually, no roentgenogram is made at this time. After reduction, abduction is reduced to about 120° so as to facilitate insertion of the nail. An incision approximately four inches in length is now made over the lateral aspect of the greater trochanter and upper two inches of the shaft exposing the lower half of the greater trochanter and upper two inches of the shaft exposing the lower half of the greater trochanter and upper one

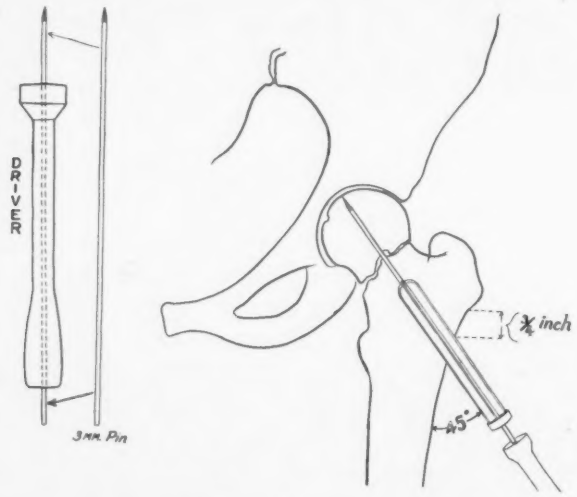

FIG. 1.—Graphic illustration of Johannson method of securing proper direction of nail. The author's tunnel driver greatly facilitates application.

or two inches of the shaft. A 3 Mm. steel wire peg is then placed into an ordinary chuck drill, and the sharp end of the wire inserted into the center of the shaft about three-quarters of an inch below the trochanter. Great care must be taken that the wire drill makes an angle with the shaft of about 45° and is angulated forward about 10 to 15° to conform to the normal anterior deviation of the neck. The wire is now drilled about two inches in this direction, after which roentgenograms are made in both planes as above described, which will confirm reduction and the exact direction of the wire drill. If reduction has not been accomplished, a second attempt can be made, but this has not been necessary in any case; also if the wire is not accurately placed, other attempts can be made until the desired direction is obtained. If reduction and direction of the wire is satisfactory, the wire is drilled into the head of the bone to within one-quarter to one-half inch of the joint, depending on the length of the proximal fragment. This is confirmed by the

roentgenogram. The protruding portion of the wire is now measured by placing a wire drill of the same length parallel with the protruding portion,

FIG. 2.—(Case 1.) Nail in position, firmly securing fragments.

FIG. 3.—(Case 1.) Solid bony union after removal of nail. Note a small piece of metal remaining in the head, a portion of the end of the tunnel. This complication should not occur if heavier material is used, as modified by the author.

and a nail of exact length selected. I employ a modification of the three-flanged Smith-Petersen nail with a steel tunnel through the center, which is slightly larger than the wire. The nail is then threaded on the wire. A driver

FIG. 4.—(Case 2.) Anteroposterior view, nail in position.

FIG. 5.—(Case 2.) Lateral view, nail in position.

with a tunnel in the center is now threaded over the wire and the nail driven through the neck and head by the aid of a heavy mallet (Fig. 1). The wire is removed and the driver placed over the trochanter, and the instrument

tapped a few times with a mallet so as to closely approximate the fragments. Roentgenograms are now made to confirm the position and the wound closed. The hip can be freely moved in the socket. Roentgenograms should not consume more than 15 minutes, and should be developed and returned to the

FIG. 6.—(Case 3.) Woman, age over 80, nail in position.

FIG. 7.—(Case 3.) Firm bony union after removal of nail at the end of seven months.

operating room in five minutes. The entire procedure requires not more than 30 to 40 minutes. Unless there is complete cooperation of the roentgenologist, the procedure is unnecessarily prolonged. When this method was first adopted, we had great difficulty in securing proper direction of the guide

FIG. 8.—(Case 4.) Bending of wire in the head of the bone, which is a serious complication and will not occur if stronger material is used.

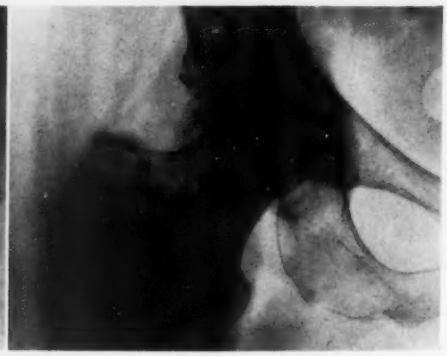

FIG. 9.—(Case 4.) End-result in Case 4 after employing the nail.

wire, but with experience, and by following, in detail, the above technic, we have become more dexterous.

No originality in the use of the nail and wire is claimed as the method was first described by Johansson,[12] of Sweden, who employed a Kirschner wire and a tunnel through the nail, but as the small flexible wire may bend

when it strikes the head, there is danger that the wire may curl itself within the head, making its removal impossible without much damage to the patient and chagrin of the surgeon, as occurred in one case as illustrated in Fig. 8.

After operation the limb is placed in a Hodgens' splint. At the end of three weeks, roentgenograms are made in order to confirm the position, which we have always found undisturbed. Motion in the knee is instituted at the end of four weeks, when the patient may be removed to his home. At the end of three months, walking is instituted without weight bearing, and at the end of four months, weight is borne on crutches. At the end of five or six months, crutches are discarded if union is firm, as demonstrated by the roentgenogram, which should show trabeculae transversing the line of fracture. Undoubtedly it is possible that function may be assumed earlier, but the physiologic status in this location is such that firm union is always slow.

FIG. 10.—(Case 5.) Apparently solid bony union but with possibly aseptic necrosis of the head. Time alone will determine the functional result.

FIG. 11.—(Case 6.) Destructive changes in the head, evidently due to extreme atrophy.

Some surgeons, as Thornton of Atlanta, do not remove the nails. Since Lane plates and other metals used for internal fixation have been known to cause complications years later, we have removed the nail at the end of six months. This can be accomplished under local anesthesia with practically no postoperative disability. The nail is loose in a high percentage of cases, and can be extracted with ease. As is well known, pressure by the metal peg usually causes bone atrophy; therefore, a circular tunnel is usually formed about the nail with an accumulation of seropurulent serum. Cultures have all been negative. In only two instances was the nail tightly fixed by surrounding bone.

This method has been applied in 35 patients, six of whom died before end-results could be determined. None of these deaths was immediately post-

operative. Seven are of less than four months' duration, and three have not returned for further observation. Nineteen have been under personal observation for a sufficient period to determine the degree of osseous union. Of these, every one has secured osseous union. The knee joints have a normal range of motion, and there is practically normal function of the hip in 17 cases. In two, even though osseous union is undoubtedly present, there have been late changes in the head. In one, there is erosion; possibly from atrophy which may have been caused by too close approximation of the nail to the articular surface. However, this patient died at the end of two and one-half years from hemiplegia and thrombocytopenic purpura. One complains of definite arthritic symptoms with pain and disability, but there is a firm osseous union. The roentgenogram demonstrates that the head is denser than the surrounding bone. This is thought to be an absolute indication that the head is dead or has become an aseptic sequestrum. However, the head of the bone may not have sufficient blood supply to undergo the normal osteoporosis that is a physiologic process in the fragments of every fracture, although there may be sufficient blood supply to keep the head alive. This has been proved by microscopic examination of some of the heads removed at operation which were thought to be dead, but were found to have a sufficient blood supply for nutrition but not for the physiologic process of callus formation, or osteoprosis.

In four cases, only ten months to one year has elapsed since treatment, but the roentgenogram and function of the limb is so conclusive of normal osseous structure of the bone and joint that little doubt remains as to the ultimate end-result. The remaining 15 have been observed for one or more years; however, all cases will be closely followed for two or more years to determine even a remote possibility of subsequent arthritic changes.

CONCLUSIONS

In conclusion it is desired to emphasize:

(1) The utter impossibility of securing accurate statistics regarding any method unless there is a differentiation as to location.

(2) That internal fixation of the neck of the femur gives by far the highest percentage of excellent functional results in the shortest period of time, which is of vast importance in elderly individuals.

(3) In only 43 per cent of fractures about the upper extremity of the femur is there difficulty in securing union, namely: Complete, central or intracapsular functions of the neck of the femur. Fifty-seven per cent are either in the region of the trochanter or impacted, in which union may be expected with rare exceptions.

(4) The presence of metal delays union, but the fixation attained apparently overcomes this objection and greatly enhances the normally delayed physiologic process.

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DISCUSSION OF THE PAPERS OF DOCTORS STRICKLER, VENABLE, AND CAMPBELL

DR. W. R. BREWSTER (New Orleans, La.).—I wish to discuss chiefly Doctor Campbell's paper on internal fixation of the neck of the femur. It was my great privilege to be associated with Dr. E. Denegre Martin in his work on internal fixation and I have followed his work since his death. His first case was operated upon in 1920 and, therefore, antedates many of the more recently reported cases and methods of internal fixation. We have been successful with it and I should say, in view of Doctor Venable's presentation, that the material used in this method has been a simple wood or hardware screw, No. 8, three inches long (sometimes three and one-half inches), and we have roentgenograms which show these screws still in position after eight years without any effect on the bones, so far as the metal used is concerned. With your permission I shall briefly review the procedure as we employ it.

Operative Procedure.—The patient is placed on the table and the leg held by an assistant in slight abduction and internal rotation of about 35°. This throws the neck of the bone on a horizontal plane. It is to be remembered that the long axis of neck of the femur is at a right angle to a plane passed through the subtrochanteric line. With the drill upon the prominence of the anteroposterior curvature of the bone, it is introduced parallel to the long axis of the neck on a horizontal plane, the drilling being continued *to* the line of fracture and not *into* the head fragment. At that time a roentgenogram is made to see if the drill is in proper position. If not it can be taken out and redirected. The drill is then removed and the screw introduced, the threads engaging the head fragment so that the head acts as a nut on a bolt and is drawn to the shaft fragment as the screw is "driven home." The first screw is not driven home but is left sticking out as a guide for the introduc-

tion of the second screw which is placed parallel to it; then both are fixed tight, the two screws preventing rotation.

We do not make it a practice to remove the screws. Roentgenograms, at times, make it appear that the screws are backing out, but that is not the case, as in subsequent ones of the same case the screws will be seen to protrude the same distance without change from year to year. Some have remained *in situ* for as long as eight years without any apparent detriment to the bone or the patient.

DR. JAMES S. SPEED (Memphis, Tenn.).—Doctor Campbell has shown that the percentage of solid bony unions obtained in central fractures of the neck of the femur has been increased from 50 to 55 per cent to approximately 80 per cent by the method of internal fixation by means of the Smith-Petersen nail. We have not as yet reached any definite conclusions as to why this marked improvement should have occurred. In the early cases treated by this method, the hip joint was exposed by a Smith-Petersen incision and completely visualized. In all these cases the Whitman manipulation was performed, and it was clearly seen that a satisfactory reduction of the fracture was obtained in practically all cases.

The same method of reduction is utilized in conjunction with the methods of internal fixation. Consequently, we have not changed our method of reduction. It is well known that in central fractures of the neck of the femur the capital fragment is deprived of its major source of blood supply. This disturbance in blood supply is also present, in the same manner, in cases treated by the Smith-Petersen nail.

Immobilization of the cases by the Whitman method in a plaster spica, while it is relatively secure, unquestionably permits a certain amount of movement at the site of fracture. It has been shown by Santos and others that aseptic necrosis of the head of the femur is not uncommon in central fractures of the neck of the femur, occurring in about 30 per cent of the cases. In those cases in which aseptic necrosis of the head occurs, nonunion results. What then has the Smith-Petersen nail added to the Whitman method? Other conditions being the same, it must be a more secure immobilization of the fracture, preventing slight movement between the fracture surfaces.

It is felt the more stable or more secure immobilization permits early growth of the capillaries across the fracture line, thus producing earlier revascularization of the head before aseptic necrosis occurs, and this is the explanation of why the results are better by means of internal fixation than by the older methods of treatment.

DR. H. EARLE CONWELL (Birmingham, Ala.).—Doctor Campbell has presented a problem which is interesting bone surgeons more, recently, than ever before. The main reason, I believe, for this interest has been on account of the poor results, such as nonunion and even mortality, which we have encountered by treating fractures of the neck of the femur by the Whitman abduction method and methods which demand bed treatment. The Fracture Committee of the American Academy of Orthopedic Surgeons is now attempting to analyze the results of the open method and internal fixation treatment of fractures of the neck of the femur. It is hoped that within the next two or three years after approximately a thousand cases have been reviewed that definite conclusions can be made concerning such type treatment.

Taken for granted that even though we will not obtain any more union in fractures of the neck of the femur by internal fixation of such fractures, I believe that it is justified because:

(1) There will no doubt be a lessened mortality.

(2) The mental attitude of the patient is entirely changed. As a matter of fact, the patient feels as if he had a few more years of normal life and activity following such an operation. It is necessary for a surgeon to see only one such instance of the mental attitude of a patient who has had internal fixation of a fractured neck of the femur to become convinced of their optimism.

(3) The economic status is improved, such as lessening of the period of hospitalization and cost of the nursing care.

(4) The disabilities about the hip and knee are seldom seen, as in the plaster spica treatment, and last, but not least, the patient is out of bed, sitting up, within a week or thereabouts following the operation.

Regarding the type of internal fixation, this depends entirely upon the method with which the surgeon is familiar.

Proper reduction is, of course, an important factor. Before the lateral view of the fractured hip became popularized I believe that we could account for at least 10 per cent of our nonunions as the result of improper reduction. I use the Leadbetter-Whitman technic in reducing these fractures. It is imperative that a lateral view always be made in a fracture of the hip before and after the reduction and operation. The lateral view can be simplified at the time of operation by flexing the thigh at a right angle to the hip as well as abducting the thigh about 15 or 20° from the midline.

Dr. John A. Caldwell (Cincinnati, Ohio).—I wish to discuss Doctor Campbell's paper because I am particularly interested in the method of open fixation. Since January, 1934, this has been our method of treatment exclusively in intracapsular fracture of the femur. During that time we have treated 50 cases. Of these 31 were of the neck, and 19 intertrochanteric. As Doctor Campbell explained, the intertrochanteric cases offer no problem so far as union is concerned. All but five cases were from the Fracture Service of the Cincinnati General Hospital, where undernourishment and alcoholism played a part in mitigating against a favorable result. Our cases have not been selected but have been taken as they came. Fourteen of the patients have died, but not as the result of the operation. One died on the day of operation from embolism, and another died two days later from pneumonia; the others died from two to four weeks postoperatively, but all died in the hospital. Twenty of the fracture of the neck cases have survived, and of those, at this time, we have five which have failed to unite. In other words, we do not feel that this open fixation is the answer to the question of union or nonunion, but it does make a great difference in the comfort of the patient and his ability to survive the ordeal that accompanies this sort of fracture. Five have not yet united; some have not been pinned long enough to say whether they will unite. We have used the Austin Moore pin, and we have adhered to the methods Speed insists upon, an accurate reduction and fixation by the pins holding the fragments as they belong. Probably the most favorable case we had was a robust, cooperative woman of about 49, who, with absolute reduction and everything apparently favorable, at the end of four months showed the head absorbing and a portion of the neck gone. While we feel that this method does make a great difference in the comfort and well being of the patient, we do not feel that we can say anything about its answering the question of union and nonunion in a fracture.

Dr. R. Arnold Griswold (Louisville, Ky.).—I think that there is no doubt but that internal fixation of the hip has come to stay, even if it does

not give a higher percentage of union. At the Louisville City Hospital, we have undernourished patients and also an admittedly inadequate nursing service. Our mortality in all fractures of the hip in patients over 50 years of age was formerly 40 per cent. Internal fixation has reduced that to ten per cent. I believe the question will be solved if we use proper mechanical and surgical principles in reduction. The fracture should be reduced in slight coxa valga, bringing the weight bearing line through the head more directly onto the shaft of the femur. The pin should be in the center of the head and as nearly parallel to the shaft as possible. This gives a straight weight bearing line along the pin and when absorption occurs in the neck, muscle pull and weight bearing cause the fragments to slide together along the pin, maintaining reduction. I am not prepared to give any statistics on the percentage of bony union. We have had three cases of nonunion, each of which can be explained by poor reduction and improper placing of the pin. We have used the method described by Doctor Campbell, but we have used from two to four wires, since I have not been able to accurately insert a single wire into the desired position every time. We pick out the most eligible wire, as determined roentgenologically, and drive the nail over that wire, pulling the others out. This method of treatment has solved many of our problems and I am sure we will continue to use it.

Dr. Frank D. Dickson (Kansas City, Mo.).—For better or for worse, the facts are: That for the past 10 or 15 years there has been a vast increase in fixation of fractures by nails, screws, and various other gadgets. The Fracture Committee of the College of Surgeons worked for several years with the Bureau of Standards in Washington to evolve bone plates and screws of a tensile strength which would reduce their breaking, which occurs often enough to be a menace, to a minimum.

No investigation, however, has been made to determine the effect of metals on tissue, particularly bone, which has arrived at any practical result until Doctor Venable carried out his research. I had the privilege of going over Doctor Venable's work at San Antonio and feel sure that he has found the answer to the question of what causes irritation about metal placed in bone, which irritation means that every screw put into a bone becomes loose at varying periods of time. You saw the evidence of irritation, in the illustrations which Doctor Venable showed, in all experiments except those in which the vitalium was used.

The practical point of Doctor Venable's paper, I think, is that he has suggested a metal which he has proved does not irritate bone and so does not become loose. This should mean that we will have available screws, nails, plates, or whatever form of fixation we wish to use to fasten bone, which will hold and give us a great deal more satisfactory and more certain fixation than we have had with metals which have been used up to this time. I think Doctor Venable's findings are very important since we may then have in our armamentarium a metal which will be nonirritating to bone.

Dr. Wm. S. Goldsmith (Atlanta, Ga.).—Like Doctor Strickler, I am a general surgeon, having a certain flair for orthopedic surgery, but today this practice is limited to acute bone injuries.

The enthusiasm following the brilliant work of Mr. Lane's method of plating long bones with metal plates and metal screws, 25 years ago, inaugurated various means of securing union of the larger long bones in cases of vicious union and nonunion in old fractures. Doctor Venable's contribution is most timely and extremely interesting.

I have a case of nonunion of the tibia in which a Lane plate, three inches long with four screws, was introduced 25 years ago. It is still in the leg of a prominent physician of Atlanta.

DR. CHARLES S. VENABLE (closing).—I probably did not make it as clear as I should have that the metals used were all different, and were employed for the purpose of creating disturbance. Among them are the vitalium screws of which I have spoken. The absence of effect on the bones is notable. There is apparently proliferation about them and not destruction. One notes, roentgenologically, the absence of reaction in the bone that was apparent with all the other screws, and the total lack of tissue reaction around the screws.

Using two steel or similar metal screws is like putting two lead plates in a battery and then trying to bring up the battery when nothing happens. Body fluids in different individuals are different in quality, which has much to do with their potency as an electrolyte. Most of the metals, in the many forms we have used previously, will cause a definite reaction due to electrolysis. This in many instances has not been sufficient to defeat the purpose, yet this reaction, in far too many instances, is so great that osteosynthesis is interrupted. The objective is to use only similar metals which are so resistant to body fluids that there will be no electrolytic force created and, therefore, no bone destruction or tissue reaction.

DR. WILLIS C. CAMPBELL (closing).—I wish to thank Doctor Speed and Doctor Dickson for bringing out certain points which were in the text of the paper but which I did not have time to discuss.

As to end-results, we have had 19 cases which we have followed through. All 19 have shown solid bony union. Two of the cases have some changes in the heads later but were solidly united. Lawson Thornton, of Atlanta, has shown the same percentage in 30 cases, and Brewster, of New Orleans, showed the same in 89 per cent, with the E. Denegre Martin technic. I do not wish to imply that end-results will always be 100 per cent, but I do believe they will be materially improved by internal fixation. Union occurs in a much shorter period of time than by the Whitman method, and if we can secure union in a shorter period of time we will have less destruction in the heads later, and a higher percentage of excellent results. There is more confusion regarding this at the present time than there was among the older surgeons who employed the terms intra- and extracapsular fractures of the neck of the femur.

In 1930, an analysis was made of the end-results of the Whitman treatment of fractures of the neck of the femur from data collected from various clinics in America. This was 26 years after the treatment was first instituted. At the present time the Fracture Committee of the American Academy of Orthopedic Surgeons is studying the end-results of treatment by internal fixation. This will not take as long as it took to determine the results of the Whitman treatment, and I believe we will soon know the relative value of the various procedures.

CONGENITAL LYMPHATIC DISEASES—LYMPHANGIOMATA

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BECAUSE of the confusion in the minds of many of the medical profession regarding many phases of the various lymphatic disorders, I have attempted a discussion of their developmental explanation, as well as a report of a group of such conditions which have come under our observation.

Just as in many other congenital disorders, if their embryology is understood, it gives the surgeon a sense of security, not only in the diagnosis but in their management. It seems an appropriate introduction to this paper to quote Dr. James E. Thompson,[1] who, in 1919, said: "Nothing can be more fascinating than a study in which truths stand suddenly revealed by a new arrangement and correlation of observations which individually are of mere academic interest. It is like finding new beauties in a picture or hidden meanings in a beloved book. Happy is the man who sees and understands, and unhappy he who is blind to the path beyond the open door, whose windows remain always barred to the universe.

"Human anatomy, viewed merely from the standpoint of bones, muscles, nerves, viscera, *etc.*, is perhaps one of the most difficult of all studies to learn and retain in one's memory. If taught without comparison between the anatomic structures of man and those of other vertebrates the study becomes a dull grind and a hopeless presentation of facts without meaning or purpose. Each structure and organ has its own history, which is inseparably connected with that of similar structures in the lower animals; and the parallel histories of these organs in each species form, as it were, a contemporary history of every species through all eternity. Viewed from this aspect, human anatomy becomes a fascinating and romantic study, in which reason is added to mere fact and purpose takes the place of coincidence."

Development of the Lymphatic System.—The present accepted theory as regards the development of the lymphatic system has been an evolutionary affair with many changes. The generally accepted facts at the present time have resulted from investigations and observations of Sabin,[3] Huntington and McClure largely. These theories are expressed briefly by quoting from Keith,[2] who says: "Recent enquiries by American embryologists, especially by Huntington and McClure, have thrown quite a new light on the origin of the lymphatic system. They have established that the formation of lymph vessels begins at definite centers and from such a center vessels spread outwards, vascularize and drain a definite area. If the starting center is excised, then there is no outgrowth and the vessels from neighboring areas invade and

drain the one thus deprived. The greatest and earliest center is situated in the angle between the jugular and subclavian veins, where the termination of the thoracic duct is afterwards formed. By the end of the eighth week the capillary network of lymph vessels was fused and formed the extensive lymph sac. In the third month outgrowths from the jugular sac on each side of the neck spread and invade the tissues of the neck, head and arm—all save the central nervous system and voluntary muscles. These are not drained by the lymphatic system. The great lymph sacs are merely tem-

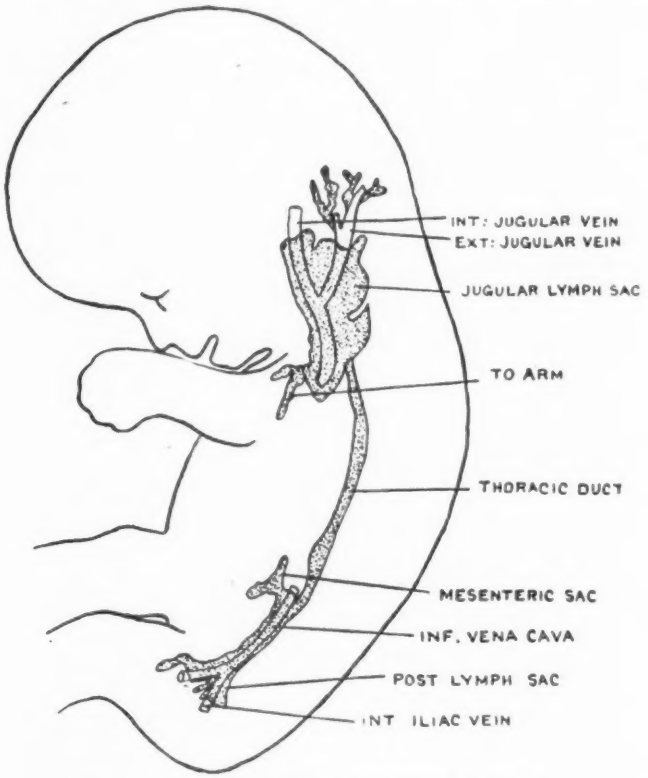

FIG. 1.—Centers of lymphatic development in embryo.

porary structures; their cavities are filled by reticular lymphoid tissue produced by the lymphatic endothelium which lines the sacs. As soon as formed, the jugular lymph sac effects a union with the jugular vein, the orifice being guarded by valvular folds.

"Another pair of lymph sacs appear in the pelvis—related to the corresponding iliac veins, into which they are first open. From the pelvic or iliac sacs outgrowths invade the hind limbs and tissues of the pelvis and buttocks. In the mesenchymal tissue in which the dorsal aorta is embedded there appear a series of endothelial-lined lymphatic spaces, which become united and place the posterior or iliac sacs in communication with the jugular sacs. In

this way two thoracic ducts are formed at the end of the second month. Two other retroperitoneal centers appear, one at the root of the superior mesentery artery, from which arises the system of vessels which drain the

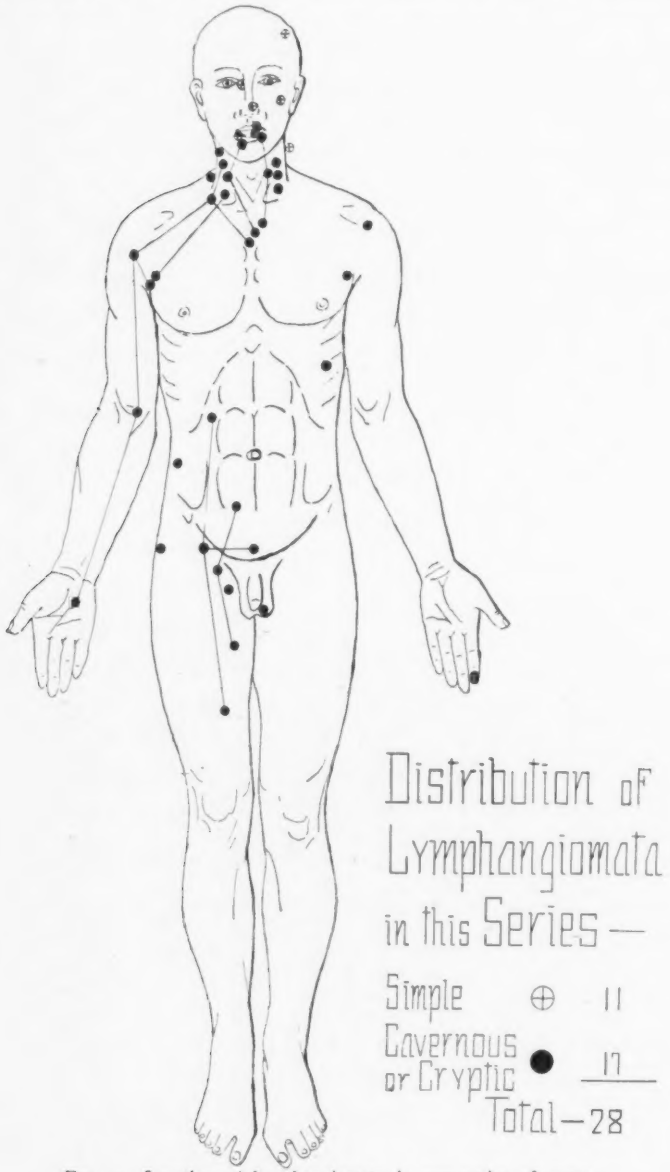

FIG. 2.—Location of lymphangiomata in our series of cases.

alimentary tract; the other, to give origin to the receptaculum chyli. The lymphatic system is just as much a 'closed' system as is the haemal system; everywhere its walls are lined with endothelium. Nowhere does it communicate with 'tissue spaces.'"

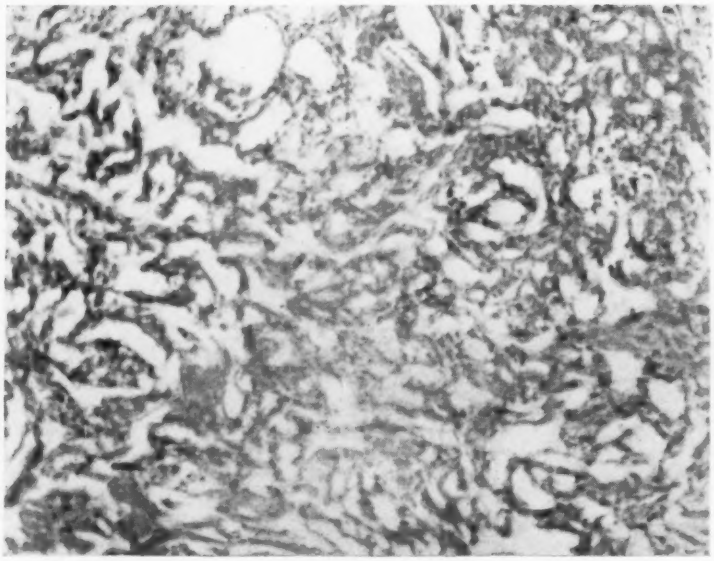

FIG. 3.—(Case 2.) Photomicrograph showing simple lymphangioma.

FIG. 4.—(Case 13.) Following radium therapy.

Sabin says: "Lymphatics are modified veins. They are vessels lined by an endothelium which is derived from the veins. They invade the body as do blood vessels, and grow into certain constant areas; their invasion of the body is, however, not complete for there are certain structures which never receive them. The lymphatic capillaries have the same relation to tissue spaces as have blood capillaries. None of the cavities of the mesoderm, such as the peritoneal cavity, the various bursae and serous cavities form any part of the lymphatic system. The lymphatic endothelium once formed is specific. Like blood vessels the lymphatics are for the most part closed vessels."

FIG. 5.—(Case 16.) Showing diffuse cavernous lymphangioma of chest wall.

FIG. 6.—(Case 16.) Photomicrograph showing cavernous lymphangioma.

With this important knowledge provided for us we are in a more advantageous position to understand and treat our clinical cases of lymphatic abnormalities.

The important lymphatic disorders to be considered may be outlined as follows:

(1) Lymphangiomata:
  (*a*) Simple or capillary.
  (*b*) Cavernous.
  (*c*) Cystic.
(2) Elephantiasis.*
(3) Chylous ascites, chylothorax, chyluria, chylopericardium, and the so called pseudochylous conditions.*

LYMPHANGIOMATA.—Generally speaking, the three varieties of lymphangiomata have a common origin.

* The consideration of Nos. 2 and 3 will be taken up in a subsequent communication.

The *Simple Lymphangiomata* are of the least importance, occurring often superficially, being easily diagnosed, and easily removed. Still, the explanation for their existence probably differs little from the other varieties.

The *Cavernous Lymphangiomata* consist of a frame work of connective tissues in which are numerous single and communicating lymphatic cysts. There are many anastomosing channels and often irregular masses of lymphocytes, lymph nodes and lymph follicles, and not infrequently one sees neoplastic blood vessels in great numbers. Where the vascularity is quite great, the term hemangiolymphangioma is applied to them. These tumors are often found in the neck, axilla, and groin, and are frequently of wide extent. Their removal may necessitate a very serious surgical procedure.

FIG. 7.—(Case 17.) Lymphangioma of scrotum.

FIG. 8.—(Case 18.) Large lymphangioma below inguinal ligament which at operation showed extension to the pelvis and retroperineally as high as the right renal vein.

The *Cystic Lymphangiomata* are those tumors with larger thin-walled convoluted cysts filled with lymph and lymphatic fluid. These forms are not completely explained by developmental disturbances alone but almost so. The most typical ones are more often found in the neck, commonly spoken of as "Cystic Hygromata." They are frequently present for a long time and then rapidly begin to enlarge. At other times after growing to a considerable size they seem to stop and rest. Keiller[4] says: "There is every reason to suppose that these cystic hygromata are at the outset cavernous lymphangiomata which become dilated with lymph, either as a result of a change in the drainage or an alteration in the function of their lining. The sudden onset and rapid growth may be explained by very active cell pro-

duction with an invasive power on the part of the endothelium and the extension of these tumors into the axilla and mediastinum has been thought to corroborate this view."

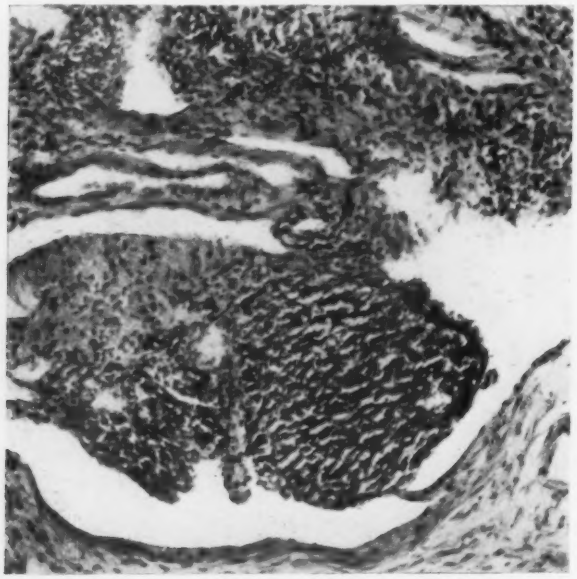

FIG. 9.—(Case 18.) Photomicrograph showing a cavernous lymphangioma.

FIG. 10.—(Case 20.) Lymphangioma of right groin.

FIG. 11.—(Case 20.) Gross specimen showing hemorrhage into cavernous space.

Therefore, wherever these centers exist congenitally, such tumors are possible, and wherever we find embryologic centers of great lymphatic pro-

duction, there we will find a greater number of such tumors, as for example in the neck, in the region of the jugular lymph sacs, subclavian, axillary, iliac and femoral, as well as mesenteric lymph sacs. If we consider these lymphangiomata as true blastomata, arising from undifferentiated mesenchyme, capable of producing lymphatic vessels by activity and growth of lymphangioblasts, in addition that there may be a blocking of outlets or possibly congenitally blind vessels, then we have sufficient cause for the responsibility of the development of these tumors. It is very probable that preformed lymphatics of normal function do not change and become in-

FIG. 12.—(Case 24.) Cystic lymphangioma.

volved in such tumor growths. Lymphangiomata, as mentioned before, are found in any part of the body, but more frequently in specific regions. Some of the most formidable, as well as interesting, are found within the abdomen, both retroperitoneally within the mesentery of the intestines, and some remarkable ones in the omentum. It is likely that few intra-abdominal cysts have any other origin than from lymphatics, if we exclude the large ovarian groups and occasionally one arising from the urogenital ridge.

Some of the outstanding reported cases are by McFadden,[5] Halsted[6] and Cohn.[7] Flynn[8] reports the removal of a large lymphatic mesenteric cyst in a young girl, 16 years of age, in which resection necessitated the removal of several feet of jejunum. Another interesting intra-abdominal

group are those found in the omentum. Girvin,[9] Outerbridge,[10] Funk,[11] Grausman and Jaffe[12] and Fisher,[13] all report interesting cases. Even as early as 1914, Outerbridge found 50 such cases in the literature. A very

FIG. 13.—(Case 26.) Cavernous lymphangioma of right cervical region.

FIG. 14.—(Case 26.) Three years after operative removal.

FIG. 15.—(Case 27.) Showing lymphangioma of right cervical region, which at operation was found to extend into the floor of the mouth and into the mediastinum.

illuminating article, with report of four cases of lymphangiomata of the neck, was written by Chas. N. Dowd,[14] in 1913. He first called attention to the relationship of the jugular lymphatic sacs of some of the vertebrate embryos with location and anatomic position of these cystic types of growth, establishing upon a firm basis the embryonic origin of these tumors.

The question of the cause of the various types of fluid which are found in lymphangiomata has been one which caused considerable controversy. It may be clear or exhibit various degrees of turbidity, particularly in the cavernous lymphangiomata. According to Keiller,[15] "This is almost certainly due to cellular contents. The fluid often contains enough albumin to coagulate spontaneously with many

cells and cell débris microscopically. In other conditions the fluid may be cloudy as a sequel to an old inflammatory process, or because of the formation of new lymphocytes. Lymph nodes are formed by the collection of lymphocytes within the lumen of the vessels and sometimes many lymphocytes are in their walls and between the lymph vessels and surrounding them. From the lymph nodes lymphocytes enter the cyst with stagnant lymph giving the milky fluid. At other times the fluid contains varying amounts of blood. This may be explained by supposing that when the lymphangioma is gradually growing and filling with lymph there may be a break down of the capillary walls with escape of blood into the sacs. At other times when

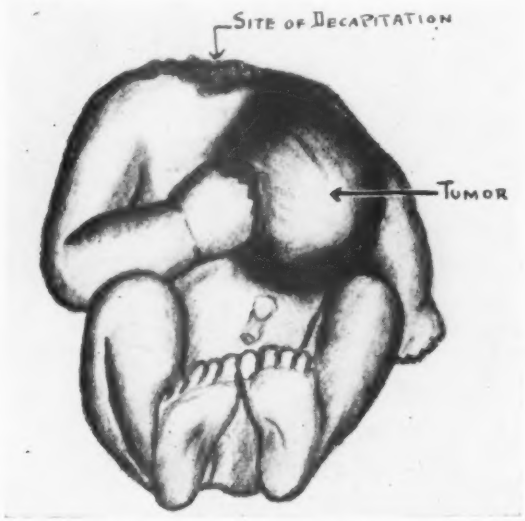

FIG. 16.—(Case 12.) Schematic drawing showing the large cystic lymphangioma in left axilla. Decapitation and puncture of the cyst was necessary before delivery could be effected.

the lymphatic vessels are small there may be a connection with the blood capillaries and lymphatic capillaries, and when the spaces are larger this communication may still be present." Both Halsted and Cohn report interesting lymphatic cysts with definite communication with large veins—the former reporting a retroperitoneal cyst communicating with the vena cava, while the latter reports an interesting lymphatic cyst of the neck definitely communicating with the jugular vein. In both of these cysts there was a large admixture of blood.

Answering a query from Doctor Halsted to Doctor Sabin in regard to this condition, the latter says: "Recent work on the lymphatic system serves to demonstrate that lymphatic vessels occur first in the neck as sacs lined with endothelium and packed with blood, which lie close to the jugular vein. The abdominal lymphatics begin as sacs which are situated close to that part of the inferior vena cava which connects the two wolffian bodies. Baetjer showed that in the pig, this sac, which is the forerunner of the retroperitoneal

lymphatics, communicates for a time with the inferior vena cava. These communications between the lymphatics and the abdominal veins, which are transitory in the pig, were shown to be permanent in the South American monkey by Silvester, while Job demonstrated similar connections in rodents. Thus the study of the development of the lymphatic system affords an explanation of anomalies involving connections between the lymphatic vessels and both the renal veins and the inferior vena cava.

Prognosis.—There are few records of the final outcome of cases not treated. Within the abdomen in the mesenteric and omental cysts intestinal obstruction is a common complication (Flynn's case). In the tongue the

FIG. 17.—(Case 28.) Cavernous and cystic lymphangioma involving the arm and hand.

FIG. 18.—(Case 28.) Following excision of cervical portion of extensive lymphangioma.

enlargement may be so great that the mouth is overfilled resulting in exposure of the tongue with drying and ulceration (case 25). The most serious complication is that of infection. Because of the dysfunction of the lymphatic chain, infection is very serious due to the rapid spreading through the connecting cysts. Also, as there are communications between the cysts and venous radicles, blood stream infection may develop. Case 19, which we report with lymphangioma in the groin, after 30 years, became ulcerated, infected and died of septicemia.

Treatment.—The treatment practiced in the past has varied. The injection of hot water into the cavernous type has been recommended by Reder.[16]

Excision is the most usual method of treatment. Some are quite easily managed but others are most difficult. The extent of the process may make their complete removal impossible (cases 18, 21 and 28). Several operations may be necessary, as recurrences are not uncommon.

The seriousness of their treatment may be appreciated when we see that of the 91 cases collected by Dowd, nearly all of which were operated upon, 38 died—a mortality of 42 per cent, the cause of death generally being infection, rarely secondary hemorrhage. Many of these cases occurred a number of years ago, when aseptic surgery was not so efficient as now, which probably resulted in a much higher mortality than exists today. Case 28 died of septicemia ten months following operation, and case 23 was septic and in a very serious condition for a long time. Two of the four cases

FIG. 19.—(Case 28.) Gross specimen of heart and mediastinum, showing large cavernous and cystic tumor of the mediastinum.

FIG. 20.—(Case 28.) Photomicrograph showing infected cavernous lymphangioma.

reported by Dowd died from sepsis following operation. At least, one should be mindful of the unusual danger of infection complicating operative procedures. The fact that infection is a much greater danger in these operative wounds than normally, because of the abnormalities of the lymphatic system, so far as I have observed, has not been referred to in the literature. Normal lymphatics have an important function in the presence of infection having the power of collecting microorganisms and filtering them out in the lymph nodes, while in lymphangiomata all lymphatic channels are not guarded by lymph nodes. Roentgen and radium therapy in certain types of lymphangiomata is highly recommended by the roentgenologist, and justly so in selected cases. In Case 25, lymphangioma of the tongue, improvement followed the use of radium. Where removal was incomplete because of wide extension within the retroperitoneal tissues and into the mediastinum, as in Case 27, which extended beneath the sternum, and which was incompletely removed, radiation apparently resulted in a cure.

TABLE I

STATISTICAL RÉSUMÉ OF 28 CASES OF LYMPHANGIOMATA

From the Surgical Service of the John Sealy Hospital, Medical Branch of the University of Texas

No.	Hosp. No.	Age	Sex	Race	Age at Appearance	Location	Treatment	Pathology	Result and Follow Up
1	636	6	M.	White	Birth	Back of neck	Excision	Simple	Recovery—no recurrence
2	8826	59	F.	White	56	Nose—near inner canthus of eye	Excision	Simple	Recovery—no recurrence
3	2979	18	F.	White	7	Tongue	Excision	Simple	Recovery and no recurrence after 2 yrs.
4	17132	29	M.	White	12	Upper lip	Excision	Simple	Recovery—no recurrence
5	10640	46	M.	White	Childhood	Buttock	Excision	Simple	Recovery—no recurrence
6	SP2261	36	M.	White	Childhood	Nose	Excision	Simple	Recovery—no recurrence
7	SP3831	—	M.	White	Childhood	Tongue	Excision	Simple	Recovery—no follow up
8	SP5592	28	F.	White	Childhood	Lower lip	Excision	Simple	Recovery—no recurrence
9	SP5846	19	F.	White	Childhood	Face	Excision	Simple	No recurrence
10	SP5850	32	F.	White	Childhood	Left index finger	Excision	Simple	No recurrence
11	SP7548	26	M.	White	Childhood	Scalp	Excision	Simple	
12	P2439	0	F.	White	Birth	Large cystic tumor size of head in the left axilla		Cavern. and cystic	The tumor was so large a decapitation and puncture of cystic tumor was necessary for delivery
13	7458	5	F.	White	Birth	Swelling in right axilla	42 mg. hrs. of radium	Cavern.	Recovery—no recurrence after 1 yr.
14	22118	7	F.	White	Infancy	Left shoulder	Excision	Cavern. and cystic	Recovery—no recurrence after 9 yrs.
15	45313	7 wks.	M.	White	Birth	Right lumbar region	Excision	Cavern.	Recovery—well 5 mos. later
16	17742	17	F.	White	Birth	Left chest wall	Excision	Cavern.	Recovery—no recurrence after 10 yrs.

17	44970	38	M.	Negro	28	Scrotum	Excision	Cavern.	Recovery—well after 2 yrs.
18	23703	18	M.	White	Birth	Right groin with extension into pelvis and upward retroperitoneally to the right diaphragm	Abdominal portion removed as high as right renal vein through right rectum incision. Complete excision below inguinal ligament	Cavern.	Recovery—no follow up longer than 1 yr. Lymphatic drainage free from wound for several weeks
19	22032	49	M.	White	Birth	Extensive tumor of right groin, lower abdomen and thigh	Radiation	Cavern. Marked ulceration and infection	Died of septicemia without operation
20	SM	3	F.	White	Birth	Right groin and upper thigh	Excision	Cavern.	Recovery—well after 2 yrs.*
21	4000	1	M.	White	Birth	Anterior and posterior triangles of neck on left	One operation 12 days. Extensive dissection with partial removal	Cavern. and cystic	Short course of roentgen therapy given 2 yrs. later. Patient well after 16 yrs. with partial facial palsy.†
22	28991	14	F.	White	Birth	Left posterior triangle of neck, rather extensive	Three operative dissections	Cavern. and cystic	Recovery—no recurrence 7 yrs. after last operation*
23	9300	8 mos.	M.	White	Birth	Extensive involvement of anterior and posterior triangles of neck on right	Extensive dissection with partial removal	Cavern. and cystic	Recovery—5 yrs. later had recurrence which was excised. Died, age 13 yrs., of pneumonia†
24	2604	5	F.	White	Birth	Anterior and posterior triangles of neck and right axilla	Excision by extensive dissection	Cavern. and cystic	Recovery—no recurrence after 2 yrs.

TABLE I (*Continued*)

No.	Hosp. No.	Age	Sex	Race	Age at Appearance	Location	Treatment	Pathology	Result and Follow Up
25	27834	35	M.	White	Birth	Lesion involved tongue, floor of mouth and right anterior triangle of neck	Excision of neck tumor followed by radium on tongue	Cavern. and cystic	Tongue ulcerated. Improvement slow. Ten yrs. later with little inconvenience
26	45858	8	F.	Negro	Birth	Right posterior triangle of neck	Excision	Cavern. and cystic	Recovery—no recurrence after 3 yrs.
27	49548	8	F.	White	Birth	Anterior triangle of neck on right with extension into mediastinum and floor of mouth	Excision, partial, followed by 1,500 units of roentgen ray	Cavern. and cystic	Recovery—no recurrence after 16 mos.
28	40930	8 mos.	M.	White	Birth	Extensive lesion involving the anterior and posterior triangles of neck, mediastinum over clavicle superficial to pectoralis major, right shoulder, axilla and arm down to hand	Excision of cervical portion	Cavern. and cystic	Ten months following the operation development of an infection in the tumor and baby died. Autopsy revealed large mediastinal lymphangioma with infection throughout tumor

* Cases of Drs. W. J. and J. L. Jinkins.
Previously reported from this Department [19] (Cases 21 and 23): ANNALS OF SURGERY, **67**, 385–396, April, 1923.

SUMMARY.—The embryologic development of the lymphatic system is discussed with an explanation of the origin of certain lymphatic tumors, which, though not truly congenital, may be considered as such. Twenty-eight cases are reported from the John Sealy Hospital, the teaching hospital of the University of Texas. The greatest danger from lymphatic tumors is infection, either with or without operation. Postoperative infection has caused many deaths. Complete removal is often impossible. Radiation is of value with or without surgery. Some are incurable.

Note: I wish to acknowledge my indebtedness to Drs. T. G. Blocker, Jr., and J. F. Pilcher, from the Departments of Surgery and Pathology, Medical Branch, University of Texas, for their assistance in compiling the data necessary for the preparation of this paper.

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DISCUSSION.—DR. ISIDORE COHN (New Orleans, La.).—The salient features of the patient upon whom I operated, and which Doctor Singleton referred to may be summarized as follows:

Case Report.—Colored, female child, age two. Mother had noticed a lump in the right side of the neck soon after birth of the child. At no time had the child experienced pain nor had there been any inflammatory manifestations.

The mass, on examination, was found to be distinctly below the angle of the jaw, it was well circumscribed, and was not adherent to the skin. The floor of the mouth was smooth and was not encroached upon by the cervical mass, which could be transilluminated.

The preoperative diagnosis was branchial cyst. At operation we found a cystic mass which overlay the internal jugular vein, within which, lying on the posterior aspect of the sac, was a second circumscribed globular bluish mass. There were tributary veins which connected this mass from within the sac to the internal jugular vein. These tributary veins were fistulous connections between the lymph cyst and the jugular vein. The cyst wall was lined by endothelium.

I believe that the endothelial lining which we found and the venous fistulae are clinical evidence which supports the observations of Sabin, McClure and others, *i.e.*, that the lymphatics of the neck have their origin from the jugular sac. At least, the case presents two of the criteria of Sabin: (1) The connection between the jugular vein and the cyst; (2) the endothelial lining.

Dr. J. Garland Sherrill (Louisville, Ky.).—It seems to me Doctor Singleton's paper has opened up a new field of investigation. I think we have all neglected this part of the anatomy and the diseases affecting it. The "why" of many of these things we do not understand. We see many tumors in the neck and the most dangerous of all types do not seem to grow for a long time and then begin to grow rapidly. Doctor Singleton has put this on a basis where we can study the "why" of these occurrences. We have always figured that the lymph always traveled along the main circuit, but we know that disease can travel backward from the proximal to the distal portion. We have a large field here and I hope the younger men will take advantage of it and clear it up much better than it has been done in the past.

Dr. James Barrett Brown (St. Louis, Mo.).—I wish to ask Doctor Singleton if he has seen any congenital lymphangiomata in association with von Recklinghausen's disease.

Dr. Albert O. Singleton (closing).—In answer to Doctor Brown's question regarding the etiology of lymphatic tissue in the tumors of von Recklingheusen's disease, I will say that this probably has a different etiology from the lymphangiomata under consideration, since it is secondary to the fibrous tissue formation in the primary neurofibromata which are the primary tumors in this disease and would be of the same character as elephantiasis secondary to the lymphatic obstruction; still the true lymphangiomata arise primarily from embryonic lymphatic centers.

FIBRO-ANGIOMA OF THE SPLEEN

REPORT OF A CASE IN AN INFANT OF FOUR MONTHS

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AS EVIDENCED by the limited number of reported cases, primary tumors of the spleen are very rare, and fibro-angiomata are extremely rare. Krumbhaar[1] and Scott, in reporting a series of 28 cases of tumors of the spleen (1928), mentioned only one hemangioma. Matas[2] states that up until 1934 there had probably been 25 cases reported as splenic angiomata, but if these reports are carefully analyzed, several of them do not conform to his five histologic requirements descriptive of a true angioma; as in several cases there was evidence of metatases to other organs resulting in death of the patient. Matas[3] cites a case of true solitary cavernous angioma of the spleen in a man 53 years old, reported by von Beckendorff. The tumor was felt two years before it was removed. It was well defined and did not invade other tissues of the body. When we consider the histology of the spleen with its large vascular spaces and its delicate trabecular structure, it is surprising that it is not a more favorable organ for the development of angioma, but this is not the case unless we assume that a large percentage of nonparasitic cysts of the spleen are secondary to angioma of earlier life. In an analysis of the reported cases this does not appear to be true. Fowler[4] carefully analyzed 82 cases of cyst of the spleen and there were only two which he thought could possibly be secondary to an angioma.

It is difficult to differentiate histologically, or clinically, between true angioma, angiomatous cysts and angiosarcoma. The diagnosis is not made before operation and the true nature of the growth is determined by microscopic study.

Matas[5] states that cavernous angiomata of the spleen present a few characteristics that have been fairly well established since their histology was first studied by Billroth and Bohm, in 1899, and by Albrecht, in 1902, *i.e.*, "(1) In the spleen as in other organs the angiomatous spaces only displace and do not replace the missing parenchyma essential to the specific function of the organ; on the other hand the endothelial lining of the vascular lacunae apparently tends to increase the physiologic function of the normal splenic pulp. (2) The structure of the splenic angiomata does not differ essentially from that of cavernous angiomata elsewhere. (3) Splenic cavernomata are supplied and nourished by a vascular system intrinsically their own and to a large extent independent of the circulation of the organ itself. (4) The angiomata of the spleen like those of the liver do not invade or permeate the splenic tissue. They may remain small, stationary, well defined and symptomless during the life of the individual unless they become cystic, then they may

attain considerable proportions. (5) They do not metastasize or spread to adjoining organs and their behavior is essentially that of benign neoplasms. When appearing conjointly with similar lesions in other organs they are evidence of constitutional angiomata and usually are hereditary." In Dowd's[6] series, published in 1915, he described such a case reported by Ernst.

Matas[7] believes that true angioma rarely metastasizes, while angiosarcoma frequently invades the liver, abdominal viscera, and even distant organs. Dowd discussed the histogenesis of cavernous angioma of the spleen in connection with his case reported in 1915, and his conclusions were that in the published cases there was not sufficient proof to justify the diagnosis of sarcoma, and stated that the cases reported as angiomata must be so regarded. He did not think they showed the characteristics of malignant tumors, but was unable to explain why these tumors at times take on unexpected and extensive growth.

The first operation of splenectomy for angioma of the spleen was performed by Martin, of Paris, in 1909, and the patient recovered. In the series reviewed by Dowd, including his case, six patients were operated upon, four recovered and two died. The seven remaining cases were reported from autopsy records. In six of the 13 cases the tumor was described as invading other organs, and if we are to assume with Matas that angiomata are benign and do not metastasize, six of the 13 reported cases were not true angiomata, but more probably sarcomata, showing secondary angiomatous change.

Case Report.—A. K., female, age four months, was admitted to Henrietta Eggleston Memorial Hospital, November 15, 1935, exhibiting symptoms of sour stomach, vomiting and the presence of a lump in the left side of her abdomen. The family history was negative except that her maternal grandmother, one uncle and three cousins had died of pulmonary tuberculosis. The past history was negative. She was a full term baby, her birth weight was 8½ pounds; she was breast fed, and in every way appeared normal until her present illness. For one month previous to admission there had been noticed a slight icteric tinge to her skin. She vomited after her feedings, was constipated, and restless.

Physical examination revealed the child to be normally developed, well nourished, but pale. She measured 24 inches in length and her weight was 12 pounds and 6 ounces. There was no lymphadenopathy and no evidence of an acute infection. In the left side of her abdomen, in the region of her left kidney, there was a firm, smooth mass which measured 8 by 5 cm. It was freely movable, being easily displaced into either the right or left lower quadrant of the abdomen. The upper and lower margins of the tumor were easily felt, as it did not extend beneath the costal margin. There was no tenderness or rigidity of the abdomen, and the liver was not palpable.

The urine was repeatedly negative except for a short while when there was one plus albumin and six to ten white blood cells. A tuberculin reaction to 0.1 mg. was obtained. Old tuberculin was negative. As shown in Table I, repeated blood studies were made and showed only a secondary anemia. The platelet count and fragility tests were within normal limits. Her temperature varied between 98.2° and 100° F. A gastro-intestinal roentgenologic study revealed that the tumor was not connected with the digestive tract; a retrograde pyelogram proved that it was not connected with the genito-urinary tract and roentgenograms of the long bones were negative for metastasis.

TABLE I

LABORATORY FINDINGS BEFORE SPLENECTOMY

	R.B.C.	Hb.	W.B.C.	Neutro.	Lymph.	Other
Nov. 15, 1935	3,140,000	70%	18,500	22%	76%	2%
Nov. 21, 1935	3,190,000	62%	10,500	30%	67%	3%
Nov. 23, 1935	Bleeding time 2½ min.					
	Coagulation time 3½ min.					
Nov. 25, 1935	3,330,000	61%	10,650	28%	68%	4%
Nov. 25, 1935	Reticulocyte count, 4.6%					
	Blood Wassermann, negative.					
	Blood Wassermann, negative.					
Nov. 25, 1936	Fragility test; hemolysis began at .46 and was complete in .36.					
	Normal hemolysis began at .48 and was complete at .36.					
Dec. 3, 1935	3,160,000	55%	17,500	64%	35%	1

A laparotomy was performed December 3, 1935. The mass was found to be a tumor of the spleen which was removed. There was no evidence of similar growth in the liver, mesentery or omentum. The postoperative course was uneventful.

The spleen weighed 70½ Gm. The body was enlarged and rounded and in this area there was a semiresistant tumor mass. On cut section the tumor was grayish-pink in color with interspersing gray areas between areas of intervening red. The tumor measured 5 by 5 cm. and was separated from the splenic tissue by a thin capsule.

FIG. 1.—Showing gross appearance of tumor in body of spleen.

Microscopic section showed the tumor to consist of proliferating fibroblastic cells which were laid down in a fairly dense compact manner. The fibroblasts varied in size and shape and grew in all directions. The blood vessels had in many areas only one or two red blood cells within them. The vessels were very numerous and there was an occasional large vascular space with a thin endothelial lining. The growth was separated from the splenic tissue proper by a capsule and the splenic tissue adjacent was flattened and atrophic.

The final diagnoses as reported by three pathologists were:

(1) Fibro-angioma. No evidence of malignancy. Prognosis good. Dr. E. L. Bishop, Atlanta, Ga.

(2) Fibro-angioma. Dr. Paul Cannon, Chicago, Ill.

(3) Fibro-hemangioma. Dr. J. C. Norris, Atlanta, Ga.

TABLE II

LABORATORY FINDINGS AFTER SPLENECTOMY

	R.B.C.	Hb.	W.B.C.	Neturo.	Lymph.	Other	Platelets
Dec. 4, 1935	3,200,000	54%	16,900	53%	56%	1%	190,000
Dec. 5, 1935	3,820,000	53%	20,300	61%	38%	1%	200,000
Dec. 6, 1935	2,980,000	54%	18,700	44%	53%	4%	210,000
Dec. 7, 1935	3,460,000	55%	22,750	52%	48%		240,000
Dec. 9, 1935	3,580,000	55%	15,700	46%	54%		300,000
Dec. 11, 1935	3,650,000	60%	12,950	42%	56%	2%	340,000

TABLE II (*Continued*)

Dec. 13, 1935	3,540,000	60%	25,500	40%	59%	1%	300,000
Dec. 16, 1935	3,900,000	61%	26,150	38%	60%	2%	550,000
Dec. 18, 1935	Bleeding time 3 min.						
	Coagulation time 3 min.						
Jan. 3, 1936	3,870,000	66%	22,150	35%	73%	2%	420,000
Feb. 25, 1936	4,900,000	68%	22,700	31%	69%		
Mar. 17, 1936	4,260,000	80%	17,200				270,000
Nov. 28, 1936	4,720,000		36,100				420,000
Nov. 28, 1936	Bleeding time 2¾ min.						
	Coagulation time 1¾ min.						

As shown in Table II, repeated blood pictures were made during the first three months following splenectomy, and there was very little change noted in the blood picture. There was some increase in the leukocyte counts but not much more than is often seen following a splenectomy in the adult. When the blood was last examined, November 28, 1936, it showed a blood picture entirely compatible with good health. The baby had shown a normal growth for one of her age, and her weight was 21 pounds.

One might well ask the question, what will be the possible effect of splenectomy in a baby four months of age? I have found no other record of splenectomy in an infant that young.

The spleen is an organ that consists predominantly of two types of tissue, one of these being reticulo-endothelium and the other being lymphoid tissue. It is estimated that the spleen contains from one-third to one-half of the lymphoid tissue of the body and approximately one-fourth of the reticulo-endothelium. Therefore, it can be assumed that the removal of a normal spleen removes a portion of these two types of tissue. There still remains, however, an adequate amount of both types to carry on their function.

The spleen is not essential to life as has been well demonstrated following its surgical removal and in congenital absence of the organ, of which anomaly Riches,[8] in 1914, collected 13 instances from the literature. He concluded that these people showed no deviation from the normal and that the absence of their spleen in no way contributed to any type of ill health. It is not an unusual finding at autopsy to find spleens in adults who have died from some totally unrelated disease, that weigh only a few grams, and, practically speaking could be considered nonfunctioning.

Wollstein and Kridel[9] performed splenectomy in 44 children, including three for traumatic rupture, 20 for rheumatic disease, one for splenomegaly, four for hemolytic icterus, eight for Cooley's anemia and eight for hemorrhagic thrombocytopenia. The ages ranged from 11 months to 12 years.

Probably the most valuable recent article on the effects of splenectomy is given in an abstract in the Journal of the American Medical Association, p. 1138, March 28, 1936, in which Ash-Upmark collected 100 cases in which the normal spleen had been removed. Ninety-nine of these were traumatic. He followed these from one to 27 years after splenectomy. In addition he collected 94 cases from the literature. He found no increased susceptibility to infections or to malignant tumors. He noted a tendency to rapid exhaus-

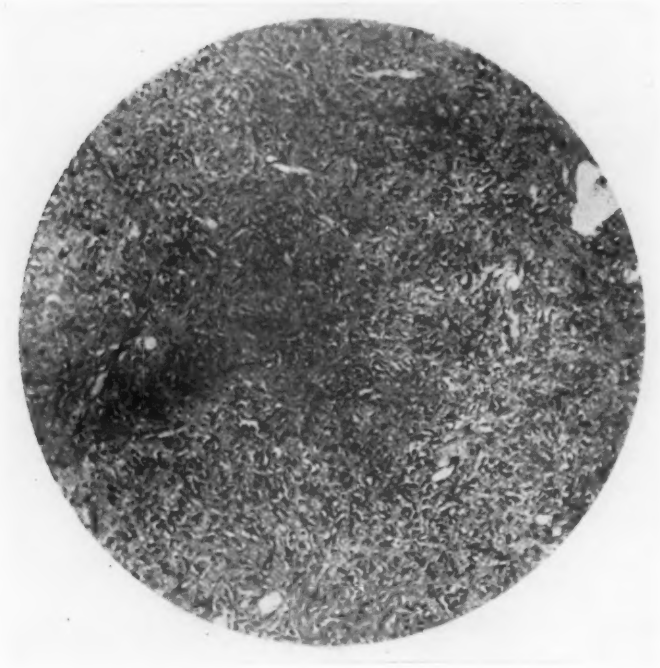

FIG. 2.—Photomicrograph of tumor (low power).

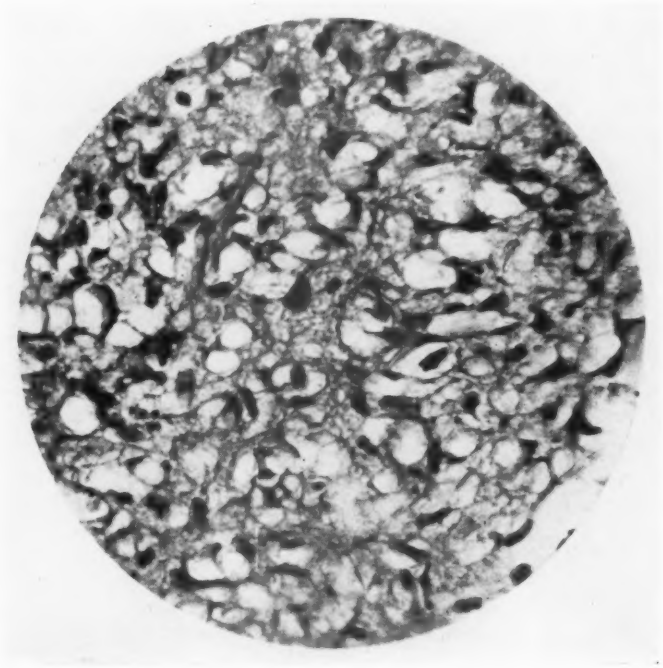

FIG. 3.—Photomicrograph of tumor (high power).

tion, and states that people whose normal spleens have been removed should be accepted for insurance on the usual terms. He concludes that: "Anatomically, physiologically and clinically, his investigations indicate that the spleen, while not necessary to life, should be removed only on vital indications."

Dominici[10] states that his results with splenectomy confirm the previous supposition that, in addition to the spleen, other sections of the endothelial system may also be responsible for excessive blood destruction, and that while splenectomy removes the principal seat of blood destruction, it does not remove all of the tissue having this function.

Kracke[11] believes that removal of the normal spleen is followed immediately by an increase in blood platelets, leukocytes and red cells. He believes further that during this postsplenectomy period the cells continue to be manufactured at their normal rate, but since part of the destructive mechanism is gone, they live to an older age in the blood stream. Therefore, it might be surmised that the average age of the cellular constituents is older than normal. This situation, however, soon corrects itself when the endothelial function is taken over by other tissues. Such compensatory hyperplasia would necessarily mean a slight generalized lymphadenopathy, which within itself would be of no importance. In the course of the lymphadenopathy, there would be excessive production of lymphocytes. Therefore, during this period, the patient may show a relative lymphocytosis.

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SUPPURATIVE ADENITIS OF THE ILIAC LYMPH NODES OF HEMOLYTIC STREPTOCOCCAL ORIGIN

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THERE have been very few reports published relative to retroperitoneal lymphadenitis during the past 15 years, and fewer still of intra-abdominal suppurative adenitis of hemolytic streptococcal origin. Although this type of infection has been recognized and studied in experimental animals, notably the guinea-pig, hemolytic Streptococcus has rarely been noted as a causal factor in intra-abdominal suppurative lesions in man. An epidemic in guinea-pigs of lymphadenitis with abscess formation due to this organism was reported by Hardenbergh,[5] in 1926. The health of the animals was not impaired to any extent, the disease being symptomless except for occurrence of the abscesses. "The mode and source of the infection was undetermined" in the animals studied. In 1929, Cunningham[4] cited an epizootic lymphadenitis in guinea-pigs due to a mucoid hemolytic Streptococcus, the disease being known as "lumps" and often characterized, in his experiments, by the formation of large retroperitoneal abscesses which had originated in the lymph nodes. As stated, the causal factors, or at least the source of infection, in these animals were not elucidated, yet it may be that some of these subjects are carriers just as certain individuals may be (Colebrook[2]). Undoubtedly in man carriers are responsible for certain cases of lymphadenitis of the intra-abdominal nodes. Colebrook found in a study of 181 normal individuals that 3 to 5 per cent showed the organism present in the nose and 5 to 10 per cent on the tonsils and walls of the nasopharynx. He thinks the hands may from time to time act as conveyors; in fact, of the 181 persons examined, 18 revealed Streptococci on the hands, in seven or 3.8 per cent of which the organism was of the hemolytic type. Five of the seven gave a history of "colds and catarrh."

In our review of the literature we have been able to find only six previously reported cases of suppurative intra-abdominal adenitis of hemolytic streptococcal origin. In Hyman's[6] review of 21 cases of retroperitoneal pelvic lymphadenitis observed at The Mt. Sinai Hospital, in the decade from 1920 to 1930, the hemolytic Streptococcus is not given as the bacterial factor. The infection in his cases occurred in the three gland groups; namely, mesenteric, lumbar and iliac, five of the 21 instances followed appendicitis, one a miscarriage, one diseased adnexa, two trauma and two infection in the lower extremities.

Primrose, in 1927, cites and discusses a case of hemolytic Streptococcus infection following operation for acute appendicitis in a young woman of seventeen. At operation there was also found a right pyosalpinx, the pus

from which later showed upon culture a growth of Streptococcus hemolyticus. The patient was treated by drainage and the intravenous injection of phenol, the latter having been shown by Maitland to increase the phagocytic action of the lymphocytes of the blood and probably also of the fixed tissue cells.

In the same year we find four additional cases published by Coutts.[3] In his series the iliac nodes alone were involved, the infection in all having its origin in the genito-urinary tract below the ureters, as one would expect if the lymph drainage of the urethra and bladder is recalled.

The above cases all made recoveries as did one observed by Topping,[9] in 1934. His case followed traumatization of the right elbow which caused a small contusion with a slight abrasion.

Recovery is not the rule in the cases with blood stream infection. Fortunately in the cases the surgeon observes the organism is arrested in the lymph nodes. These nodes belong to the hematopoietic system and in them are found lymphocytes and also monocytes. However, as the lymph flows from the afferent to the efferent lymph vessels bacteria are strained out and are here attacked by the phagocytes or macrophages formed from the fixed reticular cells of the nodes. These "energetically phagocytose varying foreign particles which come in contact with them" (Maximow). Even so, when one remembers that the abdominal lymph reaches the circulation not only by way of the thoracic and the right lymph duct but also directly by way of the venous capillaries of the follicle, thus passing directly into the blood stream (Maximow), it is difficult to understand why in such infections one does not see a general blood stream contamination. It is probable that in blood stream infections the organism may have been fed directly from the node into the circulation. In these as well as those confined to the lymphatic nodes the primary source may well be from the hands. Having shown the Streptococcus hemolyticus pyogenes to be present on the hands of 3.8 per cent of persons examined, Colebrook concludes that this accounts for the infections of fingers, open wounds and of the postpartum and postabortum uterus, believing they are probably derived from the respiratory tract via the handkerchief. In our own case the atrium of infection was a raw surface present following the rupture of a vesicle in the condition commonly called "athlete's foot."

Case Report.—G. W., male, age 24, noticed a ruptured vesicle on his left foot between the fourth and fifth toes. He painted this with mercurochrome and attended to his duties the following day. In the evening he had pain in the foot which soon extended to the lower leg and was accompanied by fever. He was examined by us the following afternoon at which time he had a temperature of 104°F., with swelling of the lymph nodes in Scarpa's triangle. The nodes were tender and much enlarged. He had had a chill. He was at once sent to the hospital from the record of which the additional data were obtained. *Chief Complaint:* Left inguinal adenitis. Ulcerative lesion left fourth interdigital space.

Present Illness.—Onset Friday, May 28, 1936. In the afternoon the patient noticed a small abrasion between the fourth and fifth toes. Also when looking downward over the inguinal region he noticed moderate swelling of the inguinal lymph nodes. He placed Whitfield's ointment over the abraded area. This did not produce any definite improve-

ment, so iodine was placed on the lesion, which caused more extensive ulceration. Also slight induration was present about the subcutaneous tissue under the fifth toe. The following morning the lymph nodes had become more enlarged and slightly tender. During the afternoon his temperature rose to 104° F., followed by a chill in spite of the application of ice packs over the inguinal region. The day of admission his temperature rose to 104° F., with three chills of such intensity as to shake the bed.

His family and past personal history were negative, as were the findings of his genitourinary, cardiorespiratory and nervous systems. There was some slight nausea.

Physical Examination.—Temperature 103° F., respiration 20, pulse 120, blood pressure 120/80. He was well developed and well nourished. Skin: Slight abrasion between the fourth and fifth toes. Lesion is weeping. Only ulcerated connective tissue present between these two toes. No evidence of lymphangitis. Inguinal nodes in the left groin are markedly swollen, slightly indurated and tender. Eyes, ears, nose and throat were negative except for pupils of eye, which were dilated. (Incidentally this dilatation was present until the latter part of his illness.) Heart, lungs, abdomen and reflexes were normal. Agglutination tests for the Bacillus of tularensis were negative as were blood cultures.

The following day there was marked tenderness on deep pressure over the lower abdomen to the left of the umbilicus and internal to the pelvic brim. Two days later tenderness extended backwards toward the costal angle and the deep lymph nodes were palpable above Poupart's ligament. At this time there was no abnormal fulness observed. On account of the high, persistent fever, blood cultures were repeatedly made. Fragility tests were normal.

On the sixteenth day radiotherapy was begun (P.K.V. 190. Filtration 0.5 Mm. copper, 1.0 Mm. aluminum. R.U. 420. Total 140 per dose. Erythema dosage 40 per cent). This was repeated on the eighteenth and twentieth days. This was followed by an apparent extension of induration toward the flank. On the thirtieth day of his illness, while the mass was smaller, there was definite point tenderness over the mass, and on July 1, he was operated upon.

Operation.—A three inch incision was made about one inch above and parallel to the crest of the ileum, midway between the anterior and posterior superior spine. The fibers of the external oblique were split and the internal oblique muscle and transversalis cut across. The peritoneum was pushed forward and an abscess containing about two ounces of pus was opened. This abscess apparently began in the deep glands around the bifurcation of the common iliac vessel and the pulsation of the artery could be felt when the finger was inserted deep into the abscess. A very soft rubber tube was placed in the abscess cavity which was further packed loosely with gauze.

On July 9, it was determined to make a further exploration higher up and posteriorly. A three inch incision was made in the left kidney region through the muscles, and a finger inserted into the perirenal space. No pus obtained but the fat and all the tissues felt definitely infiltrated. The anterior wound was then explored and an abscess cavity was found to extend backward toward this incision. The wound was packed with dry gauze. Further exploration was not made because of the danger of opening the peritoneum posteriorly.

In the meantime repeated whole blood transfusions had been given and blood cultures repeated. On the tenth day following the second exploration 1 cc. of aqueous metaphen was added to the blood given, and was followed by a decline in temperature and pulse. Three days later another 1 cc. ampule of metaphen was given intravenously and his improvement became definite and continuous thereafter until his discharge from the hospital, 11 weeks after his admission. The pulse remained quite rapid following the least exertion for a month after discharge, but at present he is perfectly well.

Critique.—During the course of the infection the following symptoms and physical signs were noted: Swelling and tenderness of and over the lymph nodes in Scarpa's triangle and later above Poupart's ligament. The tenderness was marked throughout and persisted until convalescence. As the disease progressed in the early stage this tenderness extended laterally into the flank and over the left renal region, which before the evacuation of the abscess was also quite painful to "hammer" percussion.

Before the suppuration was definitely located there was also some bulging in the ileocostal space, which was most noticeable in the lateral decubitus and over which fulness there was finger point tenderness. At no time was any evidence of the location of a suppurating node elicited by rectal examination. Once there was a slight yellowish discoloration of the conjunctiva but no rise in the icterus index.

Profuse sweating was present from the beginning as were also a marked and persistent insomnia and an appearance of anxiety. The sweating and insomnia persisted until the temperature began to decline. Nausea and vomiting at times were present and seemed to coincide with the occurrence of the chills which he had repeatedly even after the suppurating gland was evacuated.

Rigidity of the abdomen was present and was also persistent, as was abdominal distension and very active peristalsis.

The fall in red blood cell count, the pulse and temperature range are noted in the charts.

Roentgenograms were of no value in locating the area of arrest of the infection, and the curvature of the spine as pointed out by Lipsett[7] and by Beer[1] was at no time observed. There was, however, marked psoas spasm, especially upon full extension of the leg on the affected side, and the patient lay in bed with the left leg slightly flexed.

There was rapid decline in the red cells from the beginning and this persisted throughout, notwithstanding the repeated transfusions (Table I). Blood cultures were at all times negative though repeatedly made, and utilizing various media.

From the pus obtained from the abscess a gram-positive coccus was the only organism shown in the smear which was immediately made. Later from this same pus was grown a pure culture of Streptococcus hemolyticus.

Comments on Treatment.—From the first we felt an almost irresistible urge to cut down on the nodes but were restrained by the uncertainty as to the exact location of the broken down node. Even after this node was opened and evacuated we were not sure that other nodes were not as badly involved.

Radiation treatment was given, possibly late, with the hope that the infection might clear up, or if not, that the breaking down process might be hastened. We might say here that we believe in roentgen therapy in acute infections, but that is a different thesis. At the second exploration of the flank, aside from extensive infiltration, nothing was found, and the tempera-

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TABLE I

RÉSUMÉ OF HEMATOLOGIC DATA

Blood Counts	5/30/36	6/1/36	6/6/36	6/8/36	6/15/36	6/22/36	6/26/36	7/8/36	7/20/36	7/22/36	8/16/36
Red Blood Cells	4,800,000	4,100,000	3,790,000	4,010,000	4,360,000	3,340,000	4,140,000	3,135,000	3,860,000	4,030,000	4,440,000
Hemoglobin, per cent	85%	81.3%	72%	80%	90%	90%	80%	60%	78.6%	78.6%	80%
Hemoglobin, grams		12.2	10.8	12	13.5	10.5	12	9	11.8	11.8	12
Color Index		1	.96	1	1	1	1	.96	.99	.97	.9
White Blood Cells	27,000	22,900	26,250	15,800	19,700	25,150	15,750	8,550	11,750	11,600	10,550
Neutrophiles		84	91	79	83	88	84	81	60	62	43
Eosinophiles	0	0	0	1	0	0	0	0	1	1	3
Basophiles	0	0	0	0	0	0	0	0	0	0	1
Monocytes		5	4	14	7	6	4	5	8	11	8
Myelocytes	0	1	0	0	0	0	0	0	0	0	0
Lymphocytes		6	5	5	9	6	11	14	27	24	45

ture and the red blood cell diminution persisted. Both of these latter factors seem to have been markedly influenced by the administration of metaphen, particularly the temperature (Chart 1). Possibly Primrose's phenol may have had the same effect. Possibly the improvement in our case may have been a coincidence.

TABLE II

RÉSUMÉ OF URANALYSES

	6/1/36	6/4/36	6/8/36	7/20/36	8/14/36
Appearance.....	Amber, cloudy	Cloudy	Cloudy	Clear	Cloudy
Reaction.....	Acid	Acid	Acid	Acid	Alkaline
Specific gravity....	1.007	1.007	1.015	1.020	1.022
Albumin.....	Faint trace	+	++	Heavy trace	Heavy trace
Sugar.....	0	0	0	0	0
Urobilin.....				Present	
Microscopic.....	Few bacteria renal epithelial cells	Numerous coarse granular casts, many bacteria and occas. pus cast	Coarsely and finely granular casts, many bacteria and occas. pus cast	Bacteria squamous epithelial casts	Few mucous threads, uric acid crystals
Pus Cells per cm...	Occasional	Occasional	Occasional	Occasional	10–12 per H.P.F.
Red Cells per cm...	None found	None found	Occasional	90	None found

SUMMARY.—We have reported a case of hemolytic streptococcal infection of the iliac lymph nodes, reviewing briefly the literature on such infections.

Repeated blood transfusions, roentgen therapy and evacuation of a localized suppurating lymph node apparently did not arrest the infection.

Marked improvement followed by rapid recovery seemed coincident with intravenous injection of metaphen.

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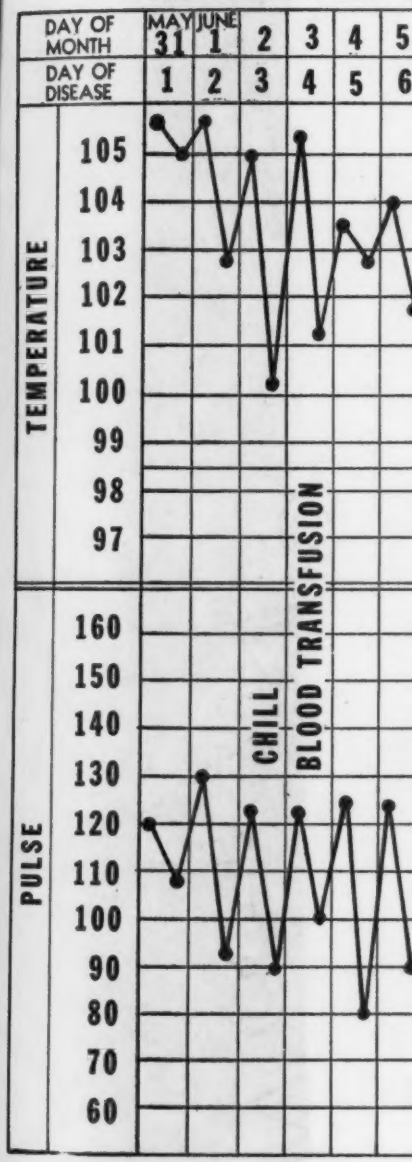
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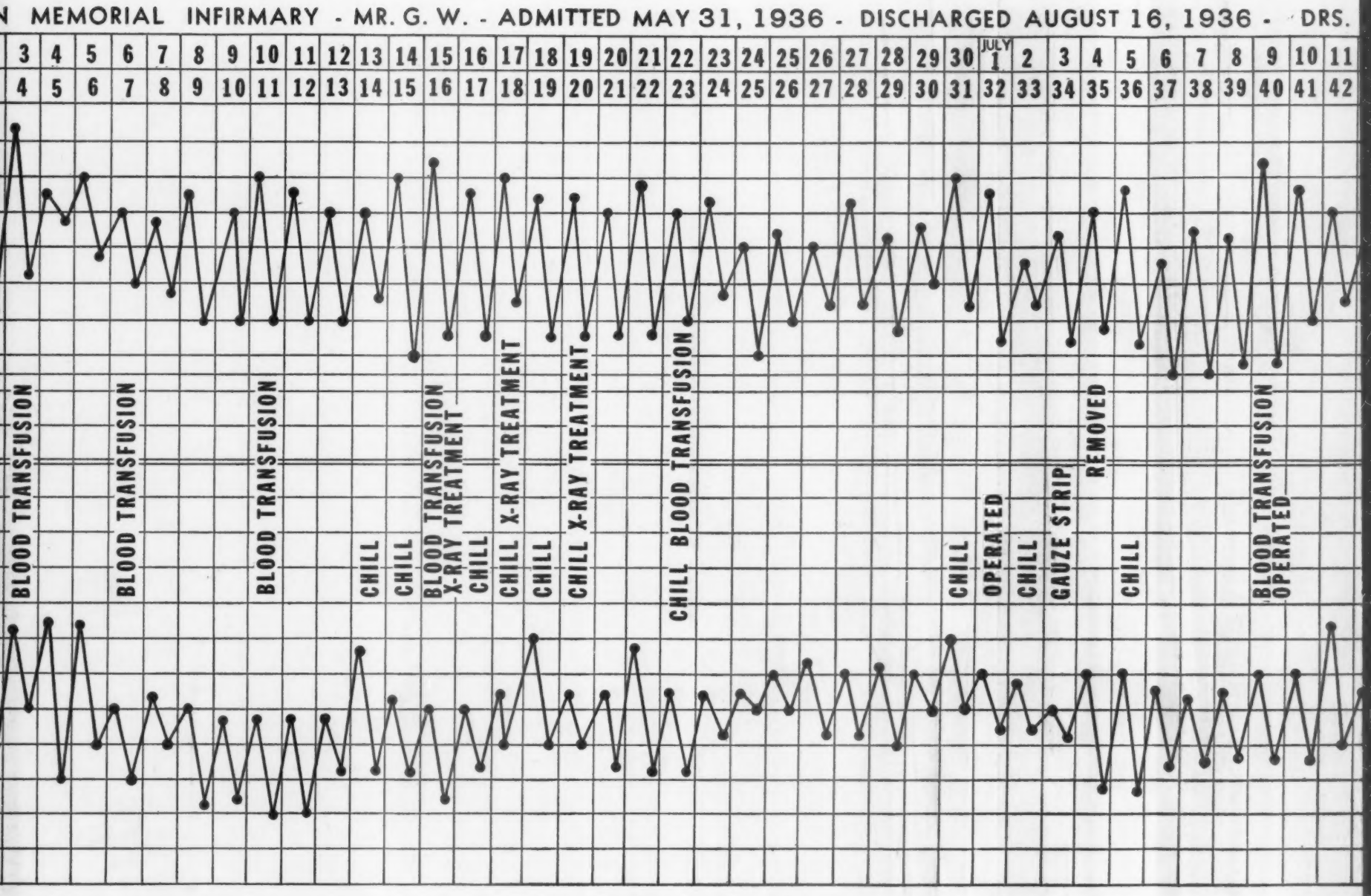
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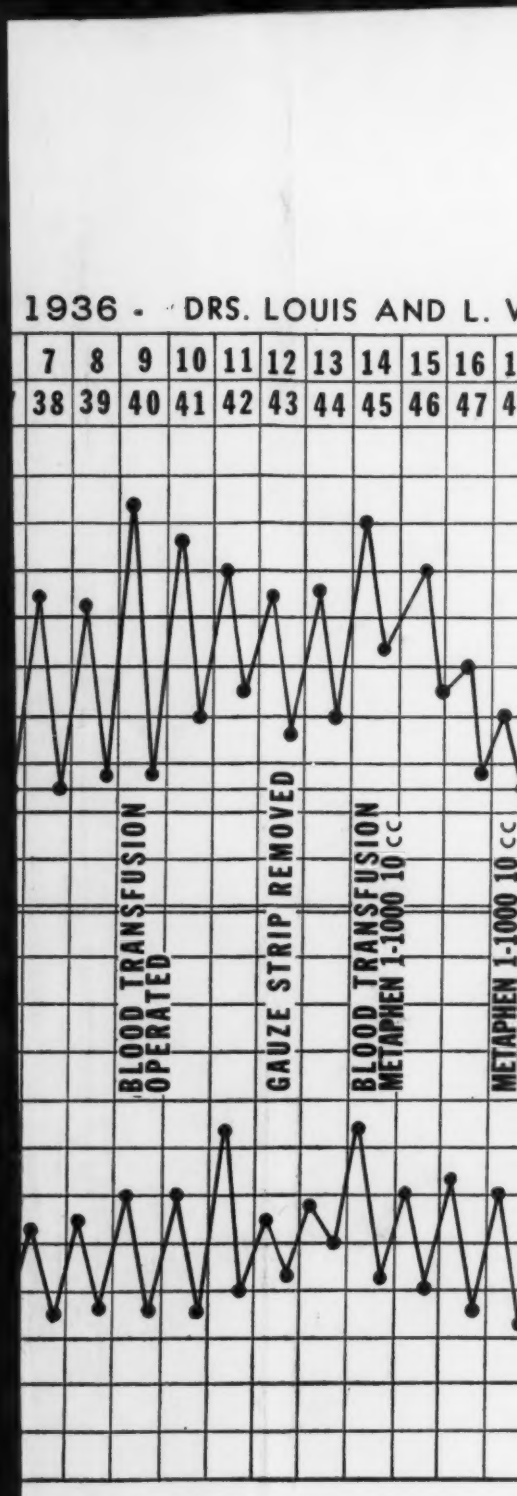
CHART 1.—Showing the fluctuations of the pulse and temperature as well as the time of the occurrence of chills, the administrat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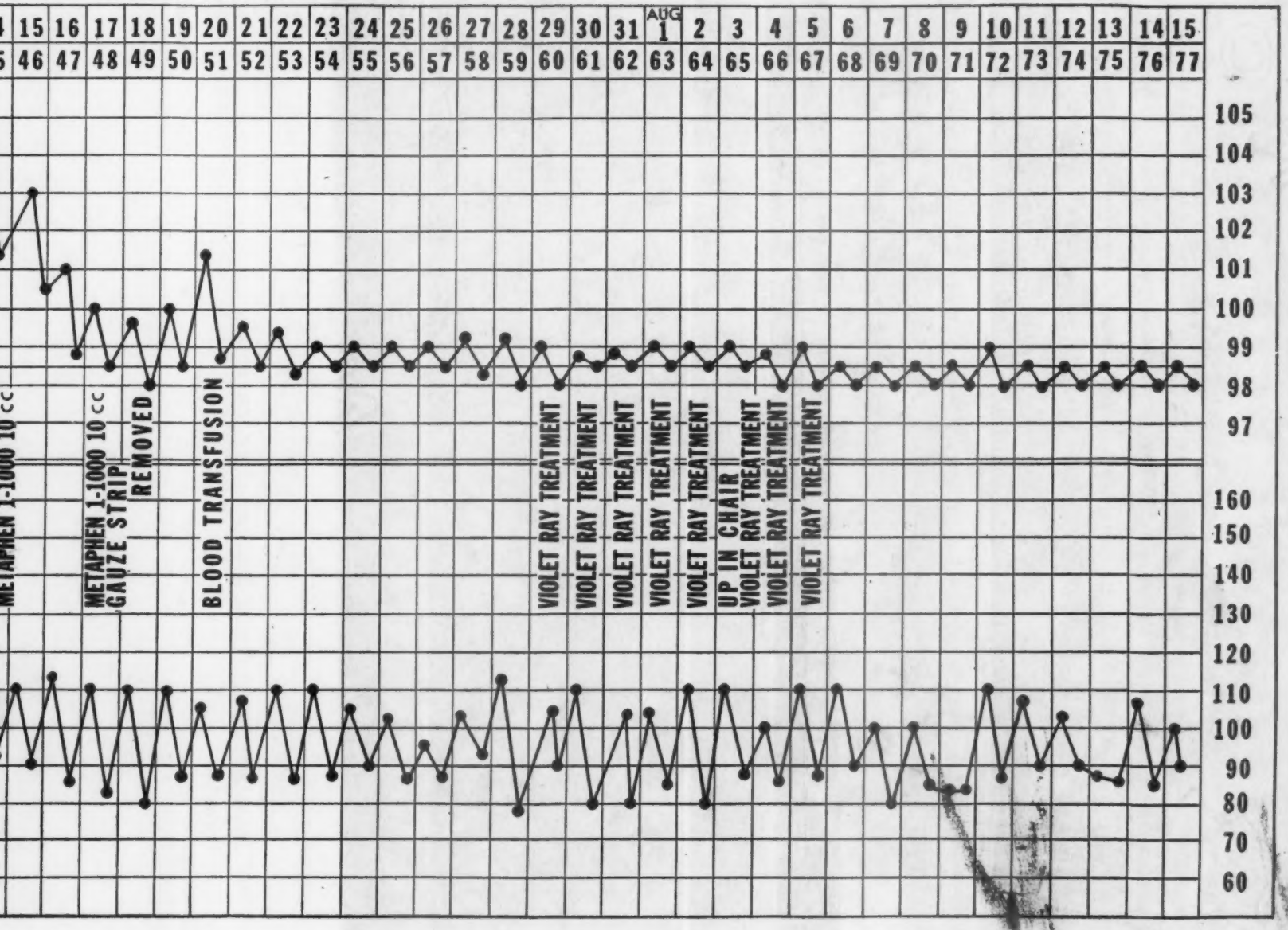
AND L. WALLACE FRANK



transfusions, roentgen therapy, operations and metaphen injections.

UNIV. OF MICH.

Discussion.—Dr. I. Mims Gage (New Orleans, La.).—Doctor Frank's paper interests me for we have had 25 or 30 cases of infection of the iliac group of lymph nodes. Most of our cases have been suppurative and most of them have been of the Streptococcus group, but not the hemolytic variety. About 5 per cent of cases will show the clinical manifestations of psoasitis, systemic manifestations of infection, and will subside without operation. We have had two cases which we thought were caused by the Streptococcus hemolyticus, but both recovered without operation. I think if one can establish infection of iliac group of nodes, it is better to wait until suppuration manifests itself before drainage is instituted. If you do not wait you are courting disaster. Recently a man of about 45 came in with a picture of suppurating iliac nodes on the right, from which we obtained about one quart of pus by incising above Poupart's ligament on the right. This healed without difficulty, but about three weeks later the patient returned with the same condition on the left side, a bilateral psoasitis. He still has a suppurating sinus on the left.

The clinical manifestations of iliac adenitis are flexion of the thigh on the abdomen, pain on extension of the thigh, and systemic manifestations of infection. If the infection is due to the Staphylococcus, the suppuration is not as severe and sometimes the abscess points below Poupart's ligament, simulating a tuberculous abscess. Incision and drainage in the fluctuating type will result in cure. In the acute type the patient usually will succumb.

THE STUDY OF WOUND HEALING

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PERHAPS an apology is due the members of the Southern Surgical Association for bringing to their attention the general subject of the study of wound healing. There was a time in the history of our profession when this subject commanded the major attention of surgeons. If they were then right in stressing the importance of the subject, certainly the vast increase in the incidence of both surgical and traumatic wounds has not lessened that importance, even though I fear that a large percentage of doctors and students of medicine have been encouraged to adopt the dangerous policy of taking for granted that the problem of wound healing has been solved.

In this audience there can be no question that the advancement and extension of modern surgery have been due more to the results of the prior studies of wound healing than to the birth of new ideas. And I, for one, am confident that a reemphasis of the principles of wound healing which were established long ago, as well as the renewal of investigative interest, especially along the lines of Curtis and others, will materially aid in improving the results of surgery as well as extending its field.

I realize that what I shall say today will neither be new nor helpful to you, but my excuse is that all the members of this association have a more or less definite responsibility for the molding and training of the lives of many who are to succeed us. With this in mind I have assumed that our viewpoint of the problem, and our efforts to teach the students and members of our graduate school of surgery the fundamental principles of wound healing and make them understand and apply them intelligently, may be of some interest to you.

In telling you of our plans, I want first to acknowledge a debt of everlasting gratitude to Dr. Wm. S. Halsted who, more than any man I ever knew, tried at all times to evaluate and apply the various principles of wound healing in his work. This was especially true in the laboratory where every study or experiment, regardless of its purpose, was, invariably, also made a problem of wound healing. For him an experiment was an unusual opportunity to observe the effects of different types of ligatures and sutures, variations of operative technic, the effects of necrosis, tight sutures, contamination, *etc.* Although the primary study was upon blood vessels, it was my duty to record most carefully the way in which the wound was handled, the character of the healing and, at subsequent operations and autopsies, to study microscopically and grossly the site of the wound. What I did not realize at the time was that he was teaching me by actual observation the significance of the various accepted principles of wound healing and was hoping that I would evaluate them and apply them to the best advantage of human beings in the hospital. With the greatest appreciation of what his time and patience

have meant to me, it has naturally become a duty in our clinic to endeavor to perpetuate his teachings; to instill the idea, so well expressed by Billroth, that "The proper treatment of wounds is to be regarded as the most important requirement for the surgeon."

For more than half a century we have been told that anesthesia, the control of hemorrhage and the control of infection have been responsible for the memorable progress of surgery. Yet we are well aware that in the life of a surgeon infection's primary rôle is a complication of the natural processes of wound healing. Even though this complication may at times be serious, tragic or even fatal, yet the fundamental processes of wound healing remain the same whether a wound heals by first, second, third or any other intention. Believing that it is erroneous to give students and young doctors the idea that the problem of wound healing is essentially synonymous with the principles of asepsis and antisepsis, we have deliberately fostered the idea that modern surgery owes its very life to anesthesia, the control of hemorrhage and to a better knowledge of wound healing. In this way the mind of the student is prepared for a consideration of all the fundamental principles involved in the problem rather than being encouraged to concentrate his whole attention on the aspect of bacterial contamination and sepsis. The development of clinical infection in a wound is by no means a matter of the presence of bacterial organisms; it is intimately related to other considerations which are also of basic importance, if one would understand the whole problem of wound healing.

Throughout the history of man's endeavor to aid wound healing, emphasis has at one time or another been placed upon various factors which are now recognized, theoretically, to be of fundamental importance. I say theoretically advisedly because it is my belief that, by and large, the doctor of this country is so dominated by the prevalent idea of asepsis and antisepsis that he is often sacrificing, to the detriment of the patient, other fundamental principles of wound healing. It is well for the student always to bear in mind that overzealousness in the utilization of any one principle of wound healing may often run counter to the application of other very valuable aids.

Among the first of the great principles of wound healing to become established was the importance of eliminating or preventing the formation of necrosis and débris in wounds. In the light of our present knowledge concerning the significance of bacteria in the healing of wounds, it has become necessary to establish in the minds of young doctors certain corollaries to this important contribution of Paré. He certainly proved that healthy living tissues have a remarkable power to ignore bacterial contamination and to combat clinical infection, even though he knew nothing about germs. Or, he proved that necrosis and débris in fresh wounds markedly handicapped their ability to handle the inevitable presence of microörganisms. And since Paré's time, it has been amply proved that necrotic tissue and foreign bodies were a definite handicap to the healing of a fresh wound even though it did not exhibit the generally accepted evidences of infection.

It is not enough to tell and show on human beings the truth of this principle of wound healing. The truth of the various principles of wound healing cannot well be driven home in the case of human beings, for the surgeon is neither willing, nor is it fair to the patient, to vary their application for purposes of teaching. And obviously it is impossible to reexamine and study the effects of any variation on human beings. Consequently, our students of surgery are naturally led into the acceptance and practice of the principles of their preceptors as a matter of mechanics secondary to surgical diagnosis and judgment. When the preceptor's basic ideas and practices are sound, then his pupil's may be as good; when they are bad, they will be perpetuated in his successors. Only when the student studies and understands the significance of the more fundamental principles of wound healing will he then be in a position to develop a technic of surgery commensurate with his abilities. In this paramount problem of surgery it is a crime to expect any student simply to perpetuate, without adequate understanding, what he has seen and helped to do.

Fortunately, traumatic, ulcerated and infected wounds, extreme fluctuations of temperatures, and laboratory experiments afford excellent opportunities for demonstrating to oneself and one's assistants the truth of the various influences upon the healing of wounds. If you make two similar so called aseptic wounds upon an animal and in one, cause a great deal of traumatism, use many ligatures and tie sutures tightly, both of the wounds may heal without apparent infection. But if those two wounds are again inspected after a week there will be a vast difference in appearance. The one with excessive traumatism and strangulating ligatures and sutures will show much white necrotic tissue (in effect, foreign bodies) while the other may show a uniform healthy granulating surface. There will be no question about which one is the better place for bacteria to grow. If many such wounds are made and with equal bacterial contamination, there is no doubt about which one will show the greatest incidence of clinical infection and suppuration. Or, to use another illustration, one can repeat the classic experiments of Halsted and others of producing peritonitis in dogs. The healthy peritoneum can withstand and overcome enormous charges of saline suspensions of bacteria, while if a piece of potato, devitalized muscle or strangulated omentum be added to the cavity, relatively small doses of bacteria will result in peritonitis.

A few visual examples of this nature afford striking demonstrations of the rôle of necrosis in wound healing and will open up for the intelligent apprentice an unlimited field of application in the practice of surgery. Above all, they will teach him that living tissues deprived of their blood supply by trauma, tight ligatures, and sutures will die even though at the completion of an operation no apparent changes can be detected in their appearance.

It is easy to observe upon human beings the value of the principle of rest in wound healing, as was formerly so convincingly done by Baron Larry,

Billroth and many others, and more recently emphasized by Orr and Koch. If one will make identical deep muscular wounds upon the extremities of two dogs and treat one dog by making him do daily walking exercise on a treadmill and the other by means of a plaster encasement, which puts the extremity at rest, there will be a striking difference in the wound healing even if there is no clinical infection. A reopening of these two wounds after one week affords an excellent illustration of the effect of trauma in the wound of the exercised dog and of rest in the wound of the immobilized extremity. Similar clinical differences in the healing of traumatic wounds as well as operative wounds of the extremities can be noted, even though the wounds are not reopened for inspection. Billroth noted this beneficial effect of rest and commented upon it as follows: "Of late I rarely see contused wounds do so well as compound fractures of the extremities, where plaster dressings are at once applied; hence we have a strong hint to compel absolute rest of an extremity with a large contused wound without fracture, by applying a fenestrated plaster splint. The cases where I have done this did remarkably well." We not only know that the immobilization lessens the reaction of wound healing, but that it also reduces materially the incidence of clinical infection. The simple conclusion is that wounds not subjected to the traumatization of activity are much more able to withstand the insults of bacterial contamination. The importance of this principle of rest should be thoroughly illustrated to, and completely understood by, every student of medicine. And it is not a difficult problem to demonstrate its effects both clinically and experimentally.

Blood Supply.—The effect of blood supply is so easily demonstrated clinically that it is not necessary to resort to animal experimentation. All that is required is to note the differences in the healing of wounds of the lower and upper extremities, and of wounds in the young and old people. Anyone who has tried to aid the healing of an ulcer in a case of peripheral vascular disease knows the importance of trying to secure for it the best blood supply. A teacher who permits his students to believe that a case of leg ulcer, when admitted to the hospital, heals because of the local applications to the wound is neglecting his duty if he does not stress the value of an improved blood supply brought about by taking the patient off his feet.

Once a medical student or apprentice surgeon really understands what blood supply means to the growth of tissue in wounds, as well as its effect upon the control of infection, there will then occur to him countless ways in which he may use this principle to aid in the healing of wounds. It is our duty to see that he does not fail to understand it.

Granulation Tissue is an inevitable concomitant of wound healing, and as such, long excited the interest and study of surgeons before there was any knowledge of bacteria. A knowledge of bacteria, and an interest in asceptic and antiseptic procedures, have not eliminated from the process of wound healing the rôle of granulation tissue. Its part in the process remains just

as important today as it did centuries ago. Do doctors and students understand that part today?

In the absence of the normal coverings and linings of the body, granulation tissue is the body's best defense against the invasion of bacteria or toxic substances. To drive home this point let the student repeat Billroth's clinical experiment which was reported as follows: "If you inject a drachm of putrid fluid into the subcutaneous cellular tissue of a dog the result will be inflammation, fever and septicemia. If you make a large granulating surface on a dog and dress it daily with charpin soaked in putrid fluid it will have no decided effect. On the border of the inflammatory new formation the lymphatic vessels are closed; on the granulating surface there are no open lymphatic vessels, hence no reabsorption takes place." Or he can make granulation tissue bleed and then note the inflammatory reaction after a dressing laden with bacteria. He can make a fresh wound by the side of a granulating wound and then dress the two with identical putrid substances and note the differences in the inflamed reaction.

If after these experiments a student begins to understand the protective and healing value of granulation tissue, he will perhaps think twice before he curettes or cuts it away, before he makes it bleed or before he permits muscular action to damage it. He may perhaps smile or know that he is being ironical when he speaks of unhealthy granulation tissue.

The principal of hemostasis in wound healing may really be considered as a subdivision of Paré's teaching concerning necrosis and débris. Blood, or blood clot, in a wound is in effect a foreign body, in addition to being doubly attractive food for bacteria. There is usually ample opportunity to demonstrate on human beings the harm to wound healing of a blood clot and also its encouragement to the development of clinical infection. This is easy to demonstrate on experimental animals.

Unfortunately strict adherence to the principle of hemostasis, by means of ligatures and cauterization, also involves a sacrifice of Paré's fundamental idea of the value of eliminating necrosis. The overzealous exponent of hemostasis may do a great deal of harm to wound healing by excessive ligation and cauterization, or the enthusiastic disciple of Paré may jeopardize wound healing by allowing hemorrhage and the formation of hematomata. The attempt to control bleeding by pressure may cause necrosis or interference with blood supply. The ideal of hemostasis which, at the present time, can be approached only from the standpoint of judgment and experience is the ligation (or cauterization) of only those bleeding points which require it, and the carrying out of this procedure in such manner as to do the least amount of tissue damage.

There is no question about the value of the principles of asepsis and antisepsis to wound healing. They are based upon the proved harm which bacteria may be to wound healing. The trouble in this country today is not due to a lack of appreciation of this fact but to the execution of aseptic and antiseptic measures in such a way as to do more harm than good by ignoring

some of the other principles of wound healing and by a failure to appreciate what living tissues themselves can do to combat the presence of bacteria. All too frequently the price of aseptic and antiseptic solutions or procedures far outweighs the harm which may have resulted from the bacteria.

It is well to let every student prove for himself the harm of one or more of the countless germicidal agents which have been advocated for use in open fresh wounds. Let him make identical wounds upon a dog, wash one with salt solution and into the other pour iodine, or bichloride of mercury or alcohol or ether, and then dress them with simple Vaseline thereafter. The inflammatory reaction will be greater and there will be more sloughing in the wound treated with the antiseptics. Besides, daily bacterial counts will show that it harbors more bacteria. Similar experiments may be carried out with skin and granulation tissue in order to teach the student what he may be doing to living tissue when he uses any substance in an attempt to kill living bacteria in or on those tissues. In this way the student will get an idea of the tolerance of the skin and granulation tissue for the various kinds and strengths of antiseptics and of the intolerance of open fresh wounds to all of them. With this fundamental background fewer students will go out into practice to use, unintelligently, all kinds of antiseptics in the treatment of wounds.

In connection with this latter point, it would be wrong to give you the impression that we believe the use of every form of antiseptic therapy is harmful; it is not wrong, however, if I convey to you our belief that the use of every antiseptic should be questioned and its possible harm to living cells should be weighed against its value in the control of infection. We admit, as does most everybody, the value of the use of antiseptics upon the skin; we decry, as I believe everybody should, their use in lieu of careful scrubbing with soap and water or their application in such strengths as to cause blistering or excoriation. Next to epithelium in point of protection, and resistance to outside insults, comes granulation tissue, upon which *only when indicated*, do we believe the mildest antiseptics may be used to advantage, and then only when their application is possible without traumatization or causing bleeding. Upon fresh wounds we believe that all known forms of antiseptic drugs or cauterizations do more harm than good. For several years it has been our custom to wash them with normal salt solution both before and after débridement and to scrub them with soap and water when necessary to remove ground-in dirt or grease. In our minds there is no question but that the wounds so treated have healed more benignly and that the incidence of clinical infection has been far less frequent. It is clear to us that inasmuch as fresh wounds cannot be made absolutely sterile, the addition of any necrosis from the use of antiseptics is a definite hindrance to wound healing.

In a very large measure, if we will adopt the principle that every wound is a problem of tissue growth, it is my belief that there will soon develop among surgeons a better understanding and appreciation of the various factors concerned in wound healing. There would be no lessening of our efforts

to reduce to a minimum the incidence of bacterial contamination, for we would know that they can destroy tissue and especially eat up the medium in which the living cells must grow. If we preached that healthy living cells have a remarkable power to kill germs, and that dead cells are helpless against their invasion, then every effort would be made to eliminate from wounds dead and devitalized tissue. We would be zealous to adapt to modern surgery the fundamental concept of Paré, especially if we thoroughly understood and taught that in every wound of every kind there are always some bacteria regardless of our effort to avoid them.

Adherence to this principle of Paré involves not only a practical effort to eliminate from wounds all visible gross necrosis but also the elimination of the use of all germicidal substances which can kill countless invisible living cells, which by their presence retard wound healing as well as encourage the growth of bacteria.

If we taught that surface traumatism as well as the buried trauma of muscular action may kill the delicate living cells of granulation tissue and also open up avenues for the spread of infection and the absorption of toxins, there would result a greater endeavor to utilize intelligently Larry's fundamental principle of rest in the healing of wounds.

If every surgeon realized that for the growth and multiplication of living cells there must be food, every effort would be made to improve and preserve the food supply. Since in the final analysis the food comes from the blood, there would be a conscious endeavor to improve the supply of blood to every wound. Efforts along this line should not be confined to securing the best circulation to a part. As we learn more about the needs of growing cells, this may require the addition of various salts and substances which may be lacking in the patient's blood. Already we know something of the value of maintaining a normal fluid balance, of the addition of glucose, salt solution and blood to the patient's blood stream. The surface has only been scratched; in this line there is still a fascinating field for investigation. Locally, too, the preservation in the granulating wound of the food or medium which has been deposited from the blood and cells is of importance. When it is not being destroyed by bacteria, the surgeon's main concern is to preserve it in the best way possible. This is mainly a matter of local noninterference and the use of dressings which will not steal it away or destroy it. When, however, the bacteria are wholly, or in a large measure, devouring it, then it becomes advisable to use such measures as will inhibit or destroy the bacteria on the granulating surface and at the same time do the least damage to the living cells which are waiting to propagate themselves. Unfortunately, most of the measures used for this purpose are also damaging to the food or medium which the cells must have for growth. Thus their use, as well as the discontinuance of their use, must be a matter of judgment based upon observation, experience and clinical teaching, as well as the support to be obtained from laboratory studies.

It is also well to bear in mind that cells have an optimum temperature for

their growth. So far as we know, this is the normal body temperature. Consideration of this influence is of particular concern in the case of surface wounds, when it is easy to observe a diminution of the rate of healing of a wound in cold weather unless efforts are made to provide a relatively normal body temperature over the surface of the wound.

CONCLUSIONS

Wound healing remains today as important as it has ever been in the history of surgery. An understanding of the processes involved is absolutely essential to the intelligent practice of surgery.

It is our duty to teach the fundamental principles of wound healing, and to put an end to the prevalent practice of permitting a student's conception of wound healing to be the result of routines of practice in busy in- and outpatient surgical clinics.

The trend of our attitude toward this problem in the past generation and a half is convincing proof that doctors, and especially surgeons, should have more knowledge of the actual processes involved in the healing of wounds. There is no justification for permitting the development of the prevalent idea that wound healing is solely a problem of asepsis and antisepsis. Bacteria are only one of several complications in the process of wound healing.

IS ADEQUATE MASKING ESSENTIAL FOR THE PATIENT'S PROTECTION?

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WITH the exception of the American farmer, there is no greater individualist than the American surgeon. It would be a waste of time for any authority, as at present constituted, to tell him that he could not operate unless he was adequately masked and expect it to be carried out, but if he can be convinced that adequate masking will protect his patient from an avoidable source of infection, then there will be no difficulty in having adequate masking adopted as a universal practice.

In hospitals with a surgeon-in-chief at the head of all surgery, it is a simple matter, if the chief so orders it, to enforce adequate masking in that institution, but ordinarily each surgeon must decide for himself on the importance of masking.

A number of articles have been written on this subject, and several years ago in a paper on "The Importance of Adequate Masking During Operation," I attempted to arouse further interest in this link in the complicated chain of operating room technic, which is so often found to be defective. Inasmuch as there are many surgeons who still operate without adequate masking, I feel that another contribution on the same subject may possibly reach some of them.

Adequate masking means the protection of the operative wound, by means of impermeable masks, from organisms which may be projected into it from either the mouth or nose of the operator or any member of the operating team.

In my work in reconstructive surgery, primary healing is particularly essential, and for this reason I have been especially interested in eliminating every avoidable source of infection.

In all well organized operating rooms, masking in some form is an almost universal practice, but this masking varies from two or three layers of loosely woven gauze over the mouth, which is practically valueless, to complete adequate masking of both nose and mouth.

With a vigorous scrub up and soaking of the hands in the solutions selected, followed by the sterile gown and intact sterile rubber gloves, the outside of which are not touched by the bare hands while being put on, we can fairly well eliminate contamination from this source. With modern, frequently checked up sterilization of all instruments, sutures, solutions and dressings which come in contact with the wound, except where there is a definite break in the technic, infection from this source can also be practically eliminated. The disinfection of the skin with any of several methods is generally effective. This leaves, roughly, two avenues from which infection

may be feared: One, from the direct discharge of microorganisms from the noses and throats of the operative personnel, and the other from the microorganisms in the air.

In studies by Meleney, Walker and others, it has been shown that serious infections of clean operative wounds have been caused by hemolytic Streptococci which were projected from the unmasked nose of the operator or some member of the operating team, and the individual responsible, and the specific organisms from the individual causing the infection have been positively identified.

In some experiments several years ago made under ordinary operating conditions utilizing always the same team, we found that there were from five to 23 more colonies grown on Petri dishes of heart muscle-blood agar, exposed for 15 minutes, when the mouths alone were masked, than when both mouths and noses were covered.

In an investigation of the bacteria of the nasal mucosa, Neumann examined over 200 individuals, of whom 111 were regarded as normal. He found diphtheroids present in 98 to 100 per cent; Staphylococcus albus in 98 per cent; Staphylococcus aureus in 30 per cent; Staphylococcus citreus and B. coli each 12 per cent; Sarcinae and pigmented micrococci each 8 per cent; Friedländer's bacillus in 6 per cent; Micrococcus roseus, pneumococcus and B. lactis aerogenes each 4 per cent; B. ozaenae and B. mesentericus each 3 per cent; molds and yeasts each 2 per cent.

When there is infection of the nasal passages other microorganisms may also be present, such as Streptococcus, hemolytic strains of Streptococcus and Staphylococcus, influenza bacillus, *etc.* It can thus be seen that the nose is the habitat of bacteria of many varieties and of varying virulence.

Meleney has found that cultures from infected clean wounds show principally white and yellow Staphylococci. Streptococci, hemolytic Staphylococci and Streptococci are grown in a much smaller number of cases, and only occasionally can other types of virulent microorganisms be cultured.

Few operators or members of operating teams know whether they are hemolytic Streptococcus or Staphylococcus carriers or not, and no one in these groups knows when he may become infected, especially in the winter epidemic season. In an interesting investigation, Meleney found that 33 per cent of the operating personnel in a New York hospital, with no epidemic going on, harbored hemolytic Streptococci in the throat and many also carried it in the nose.

In two epidemics in Boston, Walker pointed out that the percentage of Streptococcus carriers in the operating room personnel, including the surgeons, was 50 and 58 per cent respectively. We have all seen infections occur in clean cases following which a check-up was made on everything in the way of operative technic, but no attention was paid to the fact that the operator was not adequately masked. In fact, in the majority of instances no one even dared to suggest that the operator might be the source of the infection. I am convinced that many serious infections, sometimes single

and sometimes in series, which have been blamed on lack of proper sterilization of catgut, or dressings or instruments, could be traced, if properly investigated, to the unmasked nose of the operator or some member of the team.

As an example, I recently heard of an instance where a number of serious Streptococcus infections occurred in clean cases operated upon by the same surgeon. Finally, it was discovered that the surgeon was an hemolytic Streptococcus carrier and was accustomed to operating without masking his nose. This is only one of many such happenings which have come to my attention.

Sometimes infections will occur in clean operative wounds even though the entire operating personnel is properly masked, and where, as far as can be found out, there has been no break in technic. In such cases, infections by air borne microorganisms must be considered. It has been found that orderlies, who are acutely infected or are natural carriers, and who frequently pass through, or clean, the operating rooms without being masked, may be the indirect source of the infection by the air route.

Dr. Irving Walker of Boston has long been interested in the study of adequate masking and in describing the ideal mask says: "The ideal surgical mask should be one that (a) under all conditions will absolutely prevent the passage of organisms through the material of the mask in the direction of the wound, or material concerned with the operation, when both the nose and the mouth are covered; (b) will be comfortable in all degrees of temperature and will not fog the glasses; (c) will be of low original cost, or of such construction that it can be used economically many times with sterilization following each use." A mask which fulfills all of the ideal requirements has not yet been devised, but nevertheless effective masks may be made of many materials, and many types are in use.

At the present time, I use in my own work two masks made of three or four thicknesses of a closely woven muslin having from 60 to 65 strands to the square inch. Over a cap which covers the hair, one of these masks, which is sufficiently large to block out the mouth, is tied. Over this is placed a second mask which is large enough to completely cover the mouth, chin and nostrils, and this is held close to the bridge of the nose by a strip of aluminum inserted under a fairly broad piece of tape sewed across the center of the upper margin. Each mask is secured by four tapes. The layers of material do not become wet through from the breath during an ordinary operation and taken together are apparently impermeable to microorganisms. There is little if any trouble with fogging glasses, as the metal band holds the mask close to the nose, and with the use of one of the antifog preparations in addition, the glasses will remain clear.

The subject of adequate masking suggests a brief consideration of air borne bacteria, inasmuch as the bacteria projected from the mouth or nose of an inadequately masked individual in breathing, talking or coughing in the operating room, may either pass directly into a wound or subsequently may be carried into it while still suspended alive in the air or by air currents after drying.

The swing of the pendulum from the steam jet and wet technic, where every stress was laid on air borne microorganisms, to dry, and the tendency to ignore the air borne microorganisms as innocuous, has undoubtedly gone entirely too far. There is no question as to the possibility of air borne infection, and I am glad to say that much more attention is being paid to infections of clean cases caused by air borne pathogenic microorganisms.

It is well known that when an operating room has been left perfectly quiet without any one entering over a period of hours, that the dust particles in the air are less in number, and consequently the number of bacterial colonies on test plates are much fewer. On the other hand, the use of an electric fan in an operating room, which we frequently see during hot weather, stirs up every particle of dust which is in the room, and if infected material is present, which it usually is, undoubtedly serves as a most effective means of spreading it.

In Penfield's operating room in the Montreal Neurologic Institute, the air entering the room is washed with water and oil spray, but in addition in order to minimize any dust and air borne microorganisms which may still remain, the following routine is used. The operating room is cleaned in the afternoon and the floor is washed. The floor and top of the operating light, *etc.*, are covered with 50 per cent glycerin in water, which is allowed to remain so as to catch and hold any dust that may settle overnight. No one is allowed to enter the room until next morning, when final preparations are made by the nurse, who is fully gowned and masked.

Gudin, who is greatly impressed with the importance of air borne infection, reports in the Presse Médicale for March 4, 1936, an elaborate method of true air sterilization and absolute protection of the patient from this source, in specially constructed operating rooms with filtered sterilized air, air locks, *etc.*, into which only the operator, the assistant, and anesthetist are allowed to enter during the operating period. This system is apparently most effective in preventing air borne infection, but even with this set-up, unless adequate masking is practiced, the patient is still exposed to what may be the most virulent type of wound infection.

Deryl Hart has been using a method by which air borne bacteria in the operating room may be disposed of by a type of radiant energy generated in special tubes. In a personal communication, he says in regard to the radiant energy device which he uses: "It will kill a lightly sprayed culture of hemolytic Staphylococcus aureus at a distance of five feet in 60 seconds, or a heavily sprayed culture within less than five minutes at the same distance. It eliminates almost completely, within a radius of eight feet of the operating table, the shower of bacteria which can be caught on a Petri dish of blood agar at almost any time. Our postoperative results have been in keeping with what would be expected by the elimination of this rain of bacteria."

This is a very interesting and important contribution, and if prolonged exposure to this bactericidal radiant energy proves to be without harmful effect to the patient, to tissues of various kinds, or to the operating team, it

will probably be the least expensive and may be the most effective method yet devised of disposing of air borne bacteria. Even though this method may be proven 100 per cent efficient for disposing of the ordinary bacteria in the air and for hemalytic strains when exposed sufficiently long, nevertheless, adequate masking, which is required for the protection of the team from the rays, would be essential even if unnecessary for ray protection, as no rays, however effective, can destroy the vitality of microorganisms projected directly from the nose or mouth of the operator into the open wound quickly enough to prevent infection.

Eventually, it may be possible to destroy pathogenic bacteria in the noses and throats of all operating personnel by having them exposed to irradiated air of some sort, and this may in time be a valuable method of handling hemolytic Streptococcus or Staphylococcus carriers, but until this becomes an assured fact, adequate masking will still be essential.

COMMENT.—As a precautionary measure, any member of an operating team with an acute respiratory infection should be kept out of the operating room. All who are known to be carriers of hemalytic strains of Streptococcus and Staphylococcus should be banished until they are cleared of the infection.

It is significant that many of the microorganisms which are usually found in the nasal passages grow on the room control plates of operating room air. An idea of the most common microorganisms from the air which we have grown on these plates can be gathered from the following list: Staphylococcus, albus and aureus, some Streptococci; diphtheroids; Sarcinae; Micrococcus catarrhalis; B. subtilis; Proteus vulgaris; molds; a few hemolytic Streptococci and Staphylococci; a very few colon bacilli.

We have noted that the longer the time of plate exposure the greater were the number of colonies grown. For instance, room control plates when the team was completely masked with a 15 minute exposure showed 29 colonies. Under similar conditions, in the operating field, during a two hour operation 71 colonies were grown. In both of these experiments only the operating personnel were present. On the other hand, in a two hour exposure of the room, control plates in two different operating rooms with observers on the floor, one showed 96 colonies and the other 93 colonies. The presence of so many microorganisms in the air of operating rooms in a modern hospital is certainly astounding.

It is well known that patients operated upon before a crowd of observers, who are on the floor and in the stands, frequently have a stormy convalescence as far as infection is concerned, and this fact should be borne in mind by those who contemplate giving an operative surgical clinic.

In certain hospitals the observers are not allowed in the operating room at all but are in a gallery or room entirely sealed off from the operating theater. By means of reflecting mirrors, or by direct vision, or by means of opera glasses, the steps in the operation can usually be much more plainly seen than when the observer stands beside the table. The explanation, as the work proceeds, is given by the operator through a microphone and us-

ually can be clearly heard. This is of course, the safest and most satisfactory method. In the majority of operating rooms, however, observers are allowed on the operating floor or in stands near the table. It is needless to say that every observer under these conditions should be as carefully masked and gowned as the operating team, but unless this rule is continuously insisted upon it can seldom be enforced, as few observers seem to realize the danger of their bringing infection into the operating room and thence through the air to the clean wound.

Proper masking should be carried out in all accident departments, at least by the operator and his assistants. This may seem far fetched in accident rooms where air borne microorganisms is notably so numerous, but with tissues in a bruised and nonresistant state following accident, they usually have all they can do to take care of air borne contamination and contamination which clean-up or débridement cannot thoroughly remove. When to this may be added, say, hemolytic Streptococci projected from the nose and throat of the operator while carrying out the repair, there is no doubt in my mind that serious infection from this source may and does occur. If infection develops, it is of course blamed on contamination from the accident, but may well be caused by microorganisms from the nose or throat of the operator himself.

Adequate masking is just as essential for the protection of the patient during operations in every one of the surgical specialties as it is in general surgery, but many specialists do not seem to realize this.

The patient whose face is not covered during anesthetization, either general or local, should be masked when in the operating room, as there is little doubt that an unmasked patient with hemalytic strains in nose or throat may be the source of infection to his own clean wound through the air. As a matter of fact, it would be safer for us all to use an impervious mask even when doing ordinary surgical dressings.

Many times during an operation, with the patient in the usual prone position, the operator's face is directly over the wound, seldom more than 18 inches away and frequently as close as 12 inches. When we realize that microorganisms are being constantly showered from the nose into the wound during ordinary respiration, it is appalling to see an operator and his first assistant with noses uncovered breathing into an open abdomen, or an exposed brain, or a wide open breast defect, or any other wound for, say, an hour or longer. Would any of you care to be exposed to this unnecessary risk if you were the patient?

Every wound under usual operating room conditions is contaminated during operation, but fortunately relatively few become infected. Knowing what we do about the microorganisms to which clean wounds are exposed, even when adequate masking is practiced, it is extraordinary that any of them ever escape infection. That they do escape is probably due to the effectiveness of the natural resistance of the tissues and also to the fact that different individuals undoubtedly vary in their susceptibility to infection.

In my own work, I feel that an appreciable amount of contamination is avoided by the frequent washing out of the operative wound with ether, and that this procedure is largely responsible for removing many of the pathogenic air borne bacteria, which are practically always present in the operating room, as well as washing out the pabulum upon which they thrive.

Frequently, the so called filtered air of operating rooms, while somewhat less contaminated, is far from clear of dust and microorganisms. In the air conditioned operating rooms, there is little difference in the number of colonies which grow on the sterile Petri plates of blood agar. It is futile to filter and sterilize air going into an operating room when doors are constantly being opened, and attendants are passing in and out.

In some clinics, sterile boots made of some washable material are drawn over ordinary shoes or special operating shoes, and this is to be commended as it undoubtedly minimizes, to a certain degree, the amount of extraneous dust and the accompanying organisms brought in from the outside.

We all know that many operations are performed on clean cases without infection occurring, even though adequate masking is not carried out, so no one can make the statement that infection will always occur when noses and mouths are not covered. It can be stated however, without reservation, that in order to protect the patient from a source of infection, which is known to be due to inadequate masking, no surgeon is justified in exposing the wound to this avoidable hazard.

The postoperative results in those hospitals where adequate masking is a routine and where every effort is made to minimize the air borne bacteria are without question much better than where these precautions are not taken.

It is my belief that observers, cleaners, orderlies, nurses, anesthetists and doctors should be adequately masked both during the operation and whenever they come into the operating room. Every available method which has been proved of value in protecting clean cases from infection should be employed, but I am convinced that if all the accepted procedures are used in the way of clean-up and of sterilization of equipment, and of the air in the operating room by filtration or rays or any other method, it will still be essential for the operating personnel to cover their noses and mouths with impermeable masks. In short, I feel that adequate masking is not only essential but is the most important procedure, in addition to rubber gloves and gentle handling of tissues, that the surgeon can personally carry out to prevent infection in clean operative wounds.

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THE PATHOGENESIS OF LOCAL TETANUS

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MEDICAL literature is rich in historic records of many of our most commonplace diseases which have held the interest of all branches of medicine for centuries. In many instances some of these diseases have defied human intelligence and medical research, and forced the profession to realize that the day is far distant when medical science will have conquered all diseases.

Today I wish to invite your attention to the discussion of a certain phase of one of the oldest diseases known to man—tetanus—for which much has been done in a preventive way but for which the present day method of treatment is practically the same as it was many centuries past.

As a preamble to this discussion, an article from the works of Aretaeus the Cappadocian, written in the third century A.D., will serve to give you an idea of what was known of tetanus at that time and how bewildered those medical men were when called upon to treat a case of tetanus. In discussing this disease, Aretaeus (Group I[15]) wrote as follows:

"Tetanus, in all its varieties, is a spasm of an exceedingly painful nature, very swift to prove fatal, but neither easy to be removed. They are affections of the muscles and tendons about the jaws; but the illness is communicated to the whole frame, for all parts are affected sympathetically with the primary organs. There are three forms of the convulsions, namely in a straight line, backwards and forwards. Tetanus is in a direct line, when the person labouring under the distension is stretched out straight and inflexible. The contractions forwards and backwards have their appellation from the tension and the place; for that backwards we call opisthotonos; and that variety we call emprosthotonos in which the patient is bent forwards by the anterior nerves. For the Greek word *tonos* is applied both to a nerve and to signify tension.

"The causes of these complaints are many; for some are apt to supervene on the wound of a membrane, or of muscles, or of punctured nerves, when, for the most part, the patients die; for, 'spasm from a wound is fatal.' And women also suffer from this spasm after abortion; and, in this case, they seldom recover. Others are attacked with the spasm owing to a severe blow in the neck. Severe cold also sometimes proves a cause; for this reason, winter of all the seasons most especially engenders these affections; next to it, spring and autumn, but least of all summer, unless when preceded by a wound, or when any strange diseases prevail epidemically. Women are more disposed to tetanus than men, because they are of a cold temperament; but they more readily recover, because they are of a humid. With respect to the different ages, children are frequently affected, but do not often die, because the affection is familiar and akin in them; striplings are less liable to suffer, but more readily die; adults least of all, whereas old men are most subject to the disease and most apt to die; the cause of this is the frigidity and dryness of old age, and the nature of the death. But if the cold be

along with humidity, these spasmodic diseases are more innocent and attended with less danger.

"In all these varieties, then, to speak generally, there is a pain and tension of the tendons and spine, and of the muscles connected with the jaws and cheek; for they fasten the lower jaw to the upper, so that it could not easily be separated even with levers or a wedge. But if one, by forcibly separating the teeth, pour in some liquid the patients do not drink it but squirt it out, or retain it in the mouth, or it regurgitates by the nostrils; for the isthmus faucium is strongly compressed, and the tonsils being hard and tense, do not coalesce so as to propel that which is swallowed. The face is ruddy and of mixed colours, the eyes almost immovable, or are rolled about with difficulty; strong feeling of suffocation; respiration bad, distension of the arms and legs; subsultus of the muscles; the countenance variously distorted; the cheeks and lips tremulous; the jaw quivering, and the teeth rattling, and in certain rare cases even the ears are thus affected. I myself have beheld this and wondered! The urine is retained, so as to induce strong dysuria, or passes spontaneously from contraction of the bladder. These symptoms occur in each variety of the spasms.

"But there are peculiarities in each; in Tetanus there is tension in a straight line of the whole body, which is unbent and inflexible; the legs and arms are straight.

"Opisthotonos bends the patient backward, like a bow, so that the reflected head is lodged between the shoulder blades; the throat protrudes; the jaw sometimes gapes, but in some rare cases it is fixed in the upper one; respiration stertorous; the belly and chest prominent, and in these there is usually incontinence of urine; the abdomen stretched, and resonant if tapped; the arms strongly bent back in a state of extension; the legs and thighs are bent together, for the legs are bent in the opposite direction to the hams.

"But if they are bent forwards, they are protuberant at the back, the loins being extruded in a line with the back, the whole of the spine being straight; the vertex prone, the head inclining towards the chest; the lower jaw fixed upon the breast bone; the hands clapsed together, the lower extremities extended; pains intense; the voice altogether dolorous; they groan, making deep moaning. Should the mischief then seize the chest and the respiratory organs, it readily frees the patient from life; a blessing this, to himself, as being a deliverance from pains, distortion, and deformity; and a contingency less than usual to be lamented by the spectators, were he a son or a father. But should the powers of life still stand out, the respiration, although bad, being still prolonged, the patient is not only bent up into an arch, but rolled together like a ball, so that the head rests upon the knees, while the legs and back are bent forwards, so as to convey the impression of the articulation of the knee being dislocated backwards.

"An inhuman calamity! an unseemly sight! a spectacle painful even to the beholder! an incurable malady! owing to the distortion, not to be recognized by the dearest friends; and hence the prayer of the spectators, which formerly would have been reckoned not pious, now becomes good, that the patient may depart from life, as being a deliverance from the pains and unseemly evils attendant on it. But neither can the physician, though present and looking on, furnish any assistance, as regards life, relief from pain or from deformity. For if he should wish to straighten the limbs, he can only do so by cutting and breaking those of a living man. With them, then, who are overpowered by this disease, he can merely sympathise. This is the great misfortune of the physician."

A careful analysis of this article warrants the conclusion that many conditions were confused with the disease known to us as tetanus. It is evident that Aretaeus confused cases of brain injury having convulsions, as well as cases of infectious meningitis with tetanus. No mention of treatment is contained in this article, but as time goes on we find records of how cases were treated.

As late as 1828, Doctor Marsh, at the Dublin Hospital, reports several cases of tetanus, together with his treatment. It was fallacious, as is all treatment of disease where the proper knowledge of pathology is lacking. His cases were kept in vapor baths at 90° F. for five and six hours at a time, and because the moisture dissolved the crust which had formed over the original wound and made it appear cleaner, he concluded that his method was good, despite the fact that his patients all died. This is demonstrated by quotations from his article, when he says, "Notwithstanding the want of success, should we be called upon to treat other cases, we should be disposed to pursue a similar plan."

Later, in 1847, we read where cases of tetanus were purged with croton oil to eliminate toxins, and various preparations of opium used in an attempt to control convulsions. In 1848, two years after the discovery of ether as an anesthetic, an article was published by Dr. E. W. Theobald of Boston reporting the successful treatment of a case of tetanus by vapor of sulphuric ether. Following this report, ether was used rather generally, and in many instances when the initial infection was on one of the extremities, amputations were performed to rid the patient of the focus of infection. While this may seem radical, it is in line with the accepted treatment of today, when we advocate débridement in conjunction with sedatives and even anesthetics such as avertin.

Our knowledge of tetanus continued to be meager, and treatment chaotic, until a little over 30 years ago, in 1903, when Meyer and Ransom propounded their theory as to how the symptoms were produced. They believed that in cases where only a small amount of toxin is absorbed slowly, it passes up the axis-cylinder of the nearest motor nerve and affects the anterior horn cell or its connections, and local symptoms appear in the muscles supplied by those cells. When a large amount is absorbed, the toxin passes via the perineural lymphatics into the cerebrospinal fluid, and then to the central cell, as stated above, finally affecting the entire central nervous system. They explained that the jaws were first affected because the fifth nerve was short and the toxin had less distance to travel before it reached the motor cells.

Meyer and Ransom's theory of the spread of the toxin from the clostridium tetani has persisted and has continued to be the accepted theory until John J. Abel (Group I[9]) and his coworkers published the results of their experiment, in 1935, which appear far more plausible. They reported that by injecting dyes into the axis-cylinder under great pressure they were unable to force it into the central cell. They also report that by injecting

the tetanus toxin into the axis-cylinder, and not allowing any to escape into the surrounding tissues, large doses failed to produce any symptoms whatever. Nor were any symptoms produced by injecting the toxin into the perineural sheath. But when they injected small amounts of this toxin into the muscle itself, after having severed all nerve supply, they produced *local* tetanus in the muscles affected by the toxin. Larger doses involved other groups of muscles, and if sufficient amounts were injected intravenously, they produced *generalized* tetanus. Therefore, they concluded that the theory of Meyer and Ransom was incorrect. They held that symptoms are produced by the direct action of the toxin on the muscle and that the spread of the toxin was through the blood stream and lymphatics. The latter collect the toxin from the tissues, empty it into the circulation, which in turn disseminates it through the entire body and produces general tetanus. This theory is based on sound anatomic and physiologic facts and is the only plausible explanation of local tetanus.

Appended is the report of a case of local tetanus which developed as local tetanus and ran its entire course to recovery as local tetanus.

Case Report.—J. W. Mc., white, male, age 37, was first seen December 2, 1935, suffering from a laceration of the right forearm. Thirty minutes before, he had been "sideswiped" by an automobile, the door handle striking his right forearm on the lateral aspect, just below the elbow, and tearing the muscles. On examination there was a deep, ragged, transverse laceration of the lateral antibrachial muscles on the right, about one and one-half inches distal to the lateral epicondyle of the humerus. The radial nerve was exposed but uninjured.

Under 1 per cent novocain infiltration anesthesia the wound and surrounding skin were cleansed with iodine and alcohol; the muscle bellies were sutured with interrupted chromic catgut and the skin with interrupted silkworm gut. A dry sterile dressing was applied, the arm placed in a sling, and 1,500 units of tetanus antitoxin given intramuscularly into the right thigh.

The postoperative course was uneventful, the wound healing by primary union; the skin sutures were removed on the seventh day.

On the eleventh day, because of fluid underneath the skin, the skin edges were spread and a few cubic centimeters of clear straw colored fluid removed.

On December 14—12 days after the injury—the patient complained of pain in the right shoulder associated with tonic contraction of the deltoid muscle. This continued for two days, the contraction and pain becoming more marked, and on December 16—two weeks after the injury—he was admitted to the hospital.

On admission the wound was healed; the right arm was held vertical. The contraction of the deltoid could be overcome, but this caused agonizing pain. There was no trismus, opisthotonos or rigidity of any of the other muscles. The reflexes were normal and the sensorium clear.

Temperature 99° F., pulse 100, respiration 25. Blood pressure 110/70, R. B. C. 5,200,000, Hb. 90 per cent, W. B. C. 13,000 with 85 per cent polymorphonuclears, Uranalysis negative.

Two days after admission to the hospital, the contractions spread to involve the biceps, coracobrachialis and brachialis muscles, and, in addition to the vertical position of the arm, the forearm was held in extreme flexion.

Three days after admission, the wound was explored under ether anesthesia to determine the presence of any injury or pressure which might cause irritation to the motor nerves. No such conditions were found.

Four days after admission to the hospital, and 18 days after the injury, the contractions had spread to involve all the flexors of the wrist and fingers. The accompanying pain was intense and continuous.

The course of treatment can be divided into three stages:

(1) During the first four days of hospitalization the patient was given large and frequent doses of the barbiturates and salicylates with an occasional hypodermic of pantopon.

(2) During the next six days narcotics were given.

(3) Beginning on the tenth day avertin was given daily in doses of 100 mg. per kilo of body weight for a period of four days, supplemented by occasional injections of morphia or pantopon. Thereafter the patient was kept fairly comfortable with phenobarbital.

In addition to the sedatives, narcotics and avertin, a total of 69,000 units of tetanus antitoxin were given intramuscularly, none intravenously and none intrathecally.

The patient was discharged home from the hospital January 10, 1936—25 days after admission—at which time the contractions of the deltoid and upper arm had subsided. There was still moderate contraction with some pain in the flexor muscles of the forearm, the wrist and fingers being held in extreme flexion. All muscles returned to normal one month later.

Résumé.—(1) On admission pain in shoulder with continuous, *tonic* contraction of the deltoid.

(2) Contraction of the biceps ensued two days after admission.

(3) Two days later the flexors of wrist and fingers were involved.

(4) The treatment consisted of the administration of barbiturates, narcotics and avertin.

In the literature we found 17 cases reported in private practice since the World War, but every one of them finally developed in general tetanus. The case herein cited never involved any groups of muscles except those of the shoulder, arm, forearm, and hand (abduction of shoulder and flexion of arm, forearm and hand); and the only basis upon which this can be explained is the theory reported by Abel and his coworkers, which appears to have entirely disproved that of Meyer and Ransom.

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Group I comprises references relative particularly to the etiology, paths of ascent of the toxin, and types of tetanus; also the pathologic findings; Group II contains references to case reports since the War.

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DISCUSSION OF THE PAPERS OF DOCTORS HART, REID AND GREEN

Dr. Hubert A. Royster (Raleigh, N. C.)—Doctor Reid has brought out beautifully and simply the basic principles of wound treatment. The first of these is that if antiseptics are used in wounds for killing organisms, they also kill the tissue cells. For many years we have not in our service used any so called germicides in the treatment of wounds. For the past seven years we have used a 50 per cent soap solution in sterile water, really a reliable antiseptic, not only to wash the outside but also for irrigating the inside of the wound. We do this carefully with this solution and then use a soap poultice of the same type. With this bland method, wounds have healed kindly and quickly. The colored solutions of so-called antiseptics are for the purpose of dolling up and they do no good, but possibly harm. If you have a choice between a perfectly aseptic wound with dead spaces and a wound with loose sutures and proper pressure, you will invariably choose the latter. On that account we have been using in all of our clean operative wounds what we call a pressure dressing, which exerts pressure not only from above downward but from the sides. It consists of narrow pieces of

gauze in layers which are put over the incision, with short adhesive straps crossing it. Outside this we place a large dressing, in the shape of a melon slice, which is also held in position by adhesive straps; so that we not only obtain pressure from the bottom but from both sides and above. In such cases we invariably see a considerable amount of serum collect on the outside, in the gauze, instead of in the wound. This obliterates the dead spaces with proper pressure in three directions.

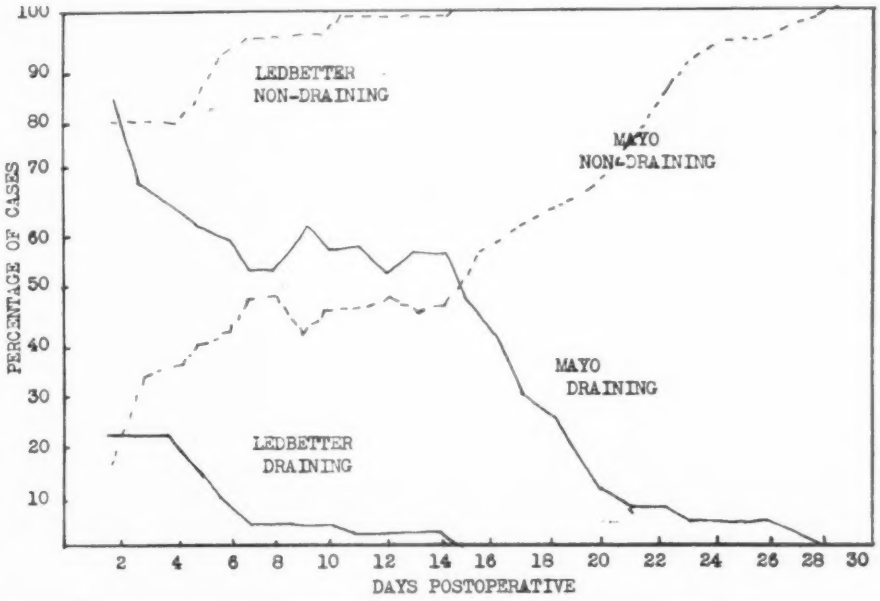
Dr. George A. Hendon (Louisville, Ky.).—Anyone who has a correct conception of antiseptics will realize at once their inefficiency. The mere matter of putting an antiseptic in a wound with the object of having it come in contact with the germs is not always tenable. You cannot kill a germ unless you catch him. To think of sterilizing a wound by antiseptics is just like painting a barn to get rid of rats. Remember, the germs are buried down in the depths of the tissue. They are hidden in the recesses and remote places, and they have the mechanism of self-defense as highly developed as you or I. Keeping that phase in view, we have devised a plan for treating septic wounds and abscess cavities, which consists of a positive pressure apparatus that operates by the electric current and which forces a stream of air through a unit that is heated by electricity. The air thus heated is forced into the wound under pressure. If one so desires, a chamber containing iodine or other antiseptic can be interposed between the heat unit and the patient. The heated air volatilizes the iodine and carries it into the depths of the wound.

Dr. Samuel L. Ledbetter, Jr. (Birmingham, Ala.).—I would like to briefly discuss Doctor Reid's paper. Of course, surgery would be impossible without proper wound healing and I think that we as surgeons can still go a little further in obtaining better results, and getting better healing of our wounds, by paying more attention to the little details and following the suggestions as outlined by him. My only wish is that all the interns and young doctors in the country could have been present and heard him. For some time I have been comparing the difference in the healing of wounds in which silk was used with wounds in which catgut was used. Also, I have been comparing the wound healing in thyroidectomies which have been drained and those that were not drained, and have come to the very definite conclusion that when silk is used and the wound not drained, wound healing is very definitely better than when we use catgut and employ drainage.

In the December 4, 1935, issue of the Proceedings of the Staff Meetings of The Mayo Clinic, Dr. H. W. K. Zellhoefer reported some very interesting observations on the wound healing of the last 50 thyroidectomies, of their 1935 series, in comparison with the last 50 cases, of their 1934 series. In this report he stated that all cases were operated upon with local anesthesia, and all, or practically all, were drained. I was interested in comparing the wound healing in his cases in which catgut was used and in which drainage was employed with our cases in which silk was used and no drainage employed. Of course, no really fair comparison could be made, due to the fact that my 50 cases represented six months' work, or a fair average of what I was doing, and the cases he analyzed were undoubtedly operated upon in a very short period of time and possibly did not represent a fair average of their wound healing, as a large percentage of these may have been very large vascular glands, substernal adenomata, *etc.*, necessitating more prolonged drainage. Chart I is an exact duplicate of his diagram upon which mine has been superimposed, and which shows very clearly the differences in the wound healing of the two technics. It will be noted that, in

Doctor Zellhoefer's series, approximately 58 per cent of his cases were still draining at the end of ten days, the average time for complete healing was 14 days, and one case drained as long as 28 days. In my last 50 cases of the 1935 series, 40, or 80 per cent, of these were closed and only ten, or 20 per cent, were drained. Of the closed cases, none drained postoperatively, the wounds were healed within 48 hours, and all dressings removed on the fifth day. Of the wounds that were drained at the time of operation, only one drained as long as 14 days (Chart I).

CHART I.



PERCENTAGE OF DRAINING AND NON-DRAINING THYROIDECTOMY WOUNDS DURING THE POSTOPERATIVE PERIOD IN THE 1935 SERIES OF 50 CASES

I think that the reason Doctor Zellhoefer's cases drained longer than those cases which I drained was due to the fact that we removed the drain after 24 hours, whereas he stated that they left them in from two to four days. Local anesthesia was also employed in my series. Chart I demonstrates very clearly the superiority of silk and nondrainage over the use of catgut and drainage.

DR. WARFIELD M. FIROR (Baltimore, Md.).—I wish to congratulate Doctor Green on bringing to the attention of this group the work of Doctor Abel on tetanus. It has been my good fortune to assist Doctor Abel in some of this work. Some four years ago Doctor Abel, in his presidential address before the American Association for the Advancement of Science, reviewed the subject of bacterial toxins. In gathering the material for this paper he was impressed with the fact that Meyer and Ransom's theory concerning tetanus toxin was out of harmony with the known facts relative to all other bacterial toxins. Doctor Abel's curiosity was aroused, and he began a series of investigations to test the validity of the theory of nerve transportation of tetanus toxin. It might be interesting to tell you of just two of our recent experiments. In order to demonstrate the existence of local tetanus, Doctor

Abel devised a method of making multiple minute injections of diluted toxin into muscles. As little as 1/200 of a lethal dose is sufficient to cause the entire hind limb of a dog to become rigid if intramuscular injections are made in 30 or 40 different places. Such a dog remains in good health and runs around with the limb fixed in extension. The rigidity is so great that one cannot alter it even under anesthesia. After three months the muscles relax and the limb returns to normal. Recently we have tried the effect of sectioning the spinal cord on the development of this type of rigidity. Regardless of whether the cord is transected before or shortly after the intramuscular injections, the limb becomes stiff and remains so. If, however, all the nerves to the limb are divided, thus interfering with the reflex arc and destroying the tone of the muscles, no stiffness results following the injection of toxin.

The second experiment that we have performed recently consisted in injecting 1/1,000 cc. of tetanus toxin into the anterior horn cells of the lumbar cord. Such an injection produces a hyperexcitable condition of the limb; the slightest sensory stimulation will precipitate a succession of forceful jerks that will last for a minute or so, until the muscles become fatigued, and then gradually subside. This type of injection does not result in a stiff leg. It reproduces, we think, the central form of the disease.

DR. ALBERT O. SINGLETON (Galveston, Tex.).—Doctor Hart's paper deserves some discussion, I am sure. At his request we ran a series of cultures in our operating room. Before doing so we felt that we were taking the usual precautions against infection, as is commonly done in most places, and we were rather surprised to find the large number of colonies following the exposure of the media for one hour on a number of days. We found that the larger the personnel in the operating room the greater the number of colonies, and seeing that certainly impresses upon one the fact that bacteria are in the air, and in prolonged operations, particularly, many of them contaminate open wounds.

I am not sure whether his method of sterilizing the air is practicable, but I am looking forward to some method which will be practicable, for I am convinced that air infection is a menace. Of course, this is not a new subject, but we had come to the conclusion that there was little danger from this source.

I visited an operating room in Copenhagen several years ago in which the floor of the amphitheater was about one inch deep in water. Everyone one the floor wore rubber boots and they were convinced that keeping down dust was very essential in lessening the bacterial content in the air.

DR. CURTIS F. BURNAM (Baltimore, Md.).—Doctor Reid has presented, in a most instructive and convincing way, the importance of increasing resistance in surgical wounds by greater care in handling and taking care of the tissues; that this will always be a fundamental principle in surgical technic is beyond doubt.

Doctor Hart has brought us something new from the other side of the picture. I think that his charts demonstrate that the so-called aseptic fever after operations is in all likelihood a septic fever. Every surgeon is familiar with the unexplained breakdown of some incisions. In addition to the wound infections, there are, now and then, some special and unusual deep seated infections which cause death. Any work directed toward removing this source of danger, provided it does not entail impossible expense, should be given the greatest consideration. Frankly, I am surprised that, using ultraviolet light, Doctor Hart is able to kill the bacteria so quickly and that the

use of radiation of this kind over an incision does not injure the tissues themselves. However, as I have had no experience personally with this matter, I think we must remember that experience is the best teacher and that preconceived ideas must be given up in the light of actual experimentation. It would seem to me that it might be possible to sterilize the air in the operating room, and to use some form of mask for the operator and his team, which would prevent any infection from their respirations.

I feel that Doctor Reid and Doctor Hart should be congratulated on the splendid work they have done and their excellent presentation of it.

DR. FRANK H. LAHEY (Boston, Mass.).—Doctor Overholt has been particularly interested in Doctor Hart's work and we are installing a similar apparatus. He has interested Doctor Wells in the department of physics at Harvard, where they are also installing an apparatus which I think may be useful. I have objected to having to operate with goggles and with my head encased. They have suggested that they put in the lights so that they throw the rays upward and this will irradiate the upper air. They think it may be possible to sterilize all the air in the operating room by exposing the upper layer of air to these rays, the motion of air eventually resulting in its all being irradiated. I do not know whether it will accomplish the same results that Doctor Hart has. We have exposed plates for various periods, and it is true that the air in all operating rooms is contaminated.

DR. J. M. T. FINNEY (Baltimore, Md.).—I, too, want to pay tribute, as did Doctor Reid, to that great surgeon-teacher, Doctor Halsted. Doctor Reid has epitomized in an admirable way the principles, not all original, to be sure, with Doctor Halsted, but the fundamental principles of good surgery for which he stood and contended throughout his entire surgical life. This was one of Doctor Halsted's outstanding contributions; namely, the scrupulous observation and practice of the fundamental principles of surgery in the gentle handling of tissues, the avoidance of trauma, the stoppage of hemorrhage, the avoidance of foreign bodies in the wound, such as tissue masses that had been strangulated by ligatures, the accurate coaptation of wound edges, and the avoidance of tight sutures. It is curious how often one sees these cardinal principles being violated by surgeons all over the country, unconsciously to be sure, in the undue hurry to get through with the operation. One will at times unconsciously violate these fundamental principles if one is not constantly on one's guard. In doing so, drainage of the wound is rendered necessary when the proper observance of these rules would make it unnecessary.

I think Doctor Hart's work is deserving of great credit. I am thoroughly in favor of any advance, but I think we should stop and think as we go along, and be sure that we ourselves are not guilty in our work, of violating those principles which have been so splendidly brought to our attention by Doctor Reid. Every surgeon knows from experience that the peritoneum will take care of infection better than any of the other tissues composing the abdominal wall; that next perhaps is the muscular tissue, and that most liable to infection is the skin and subcutaneous fatty tissue. In closing infected wounds I have for some time been using instead of skin sutures, long narrow strips, one-sixteenth or one-eighth inch wide, of flamed adhesive plaster and applying these instead of sutures to hold the skin in apposition. They should be long in order to splint, as it were, the adjoining skin. By this means we can bring and hold the tissues in as exact approximation as with sutures. We have yet to see infection in a wound where these

narrow strips of flamed adhesive have been used instead of sutures. It is a small thing, of course, but it has proved most useful.

Dr. Deryl Hart (closing).—I wish to express my indebtedness to my associates and staff for their assistance in this work, and also to those who made cultural observations for me in different parts of the country. Since there were few reports in the literature, I requested these cultures in order to confirm my impression that contamination of the air in operating rooms is quite universal, and to refute the statement that had been made that it was a local condition with us.

Referring to Doctor Burnam's discussion, it has been quite surprising to find that with such a small amount of current we could kill bacteria within a distance of eight to ten feet, and particularly that this could be done within such a short period of time. We believe that this high bactericidal effect and relatively low irritative action is due to the fact that this radiation is of a wavelength predominantly below 2,600.

As to the radiation extending upward over the operating region, as mentioned by Doctor Lahey, I hope that will work successfully, as it will simplify the matter of protection for the staff. In my opinion, the major part of the contamination of the air of the operating room comes from the personnel. The organisms given off by them will therefore be beneath the tent of rays and therefore they may get into the wound without being exposed to the radiation. This distribution of radiation may cut down the air contamination by sterilizing the air that passes within its effective range. It will not interpose an effective barrier between the wound and the source of the contamination.

I thoroughly believe in all the principles Doctor Reid brought out in his excellent paper. I do not feel, however, that we should rely upon them entirely to prevent bacterial growth in a wound if we have a method that will prevent, or greatly reduce, the inoculation of this wound. Aseptic surgery should be our ideal. Relatively atraumatic surgery should be practiced at all times, and antiseptic surgery used if the necessity arises.

Dr. Charles C. Green (closing).—I wish to bring out one interesting fact in connection with the work of Doctor Abell and his coworkers; namely, that he has proved the statement made, in 1861, by the Norwegian, Doctor Heiberg, to be correct. At that time Doctor Heiberg stated: "Tetanus is not a disease of the nervous system, but a blood disease, or blood poisoning, which has its direct effect upon the muscle tissue."

Despite this statement, however, the medical profession accepted the theory of Meyer and Ransom in 1903; but now some of the statements of Meyer and Ransom appear almost as ridiculous as the article quoted from Aretaeus the Cappadocian, as, for example: Meyer and Ransom explained the stiffness of the jaws by saying that the fifth nerve was so short that the toxins traveled through its axis-cylinder so quickly that the rigidity appeared in the muscles of the jaws first. At this time that statement appears ridiculous, but how do we know that in 25 years more the things we are saying today will not appear even more ridiculous?

THE ORIGIN AND GROWTH OF RENAL CALCULI

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FIVE thousand years before the Christian era a man died in the upper Nile Valley, and his body was prepared for burial following the custom of his day. A few years ago an English archeologist, on examining this grave, found a concretion lying among the pelvic bones, which, on analysis, was found to be composed of an outer crust consisting of calcium-magnesium-ammonium phosphate and an inner body consisting of almost pure uric acid crystals. Through the intervening seven thousand years, therefore, the chemical character of urinary calculus has remained unchanged. Likewise, the surgery of "calculus disease" enters medical history with its very beginning and, following many strange and interesting vicissitudes, comes down to us today broadly considered in all surgical writings, occupying through all time a prominent place in the surgical clinic, and hallowed by the names of all great surgeons of the past. As such it has come to be known as "calculus disease," and surgery's effort has remained little changed, interpreting its duty to be the removal of this morbid product.

It is to be part of my present effort to change this point of view, and to try to show that urinary calculus is but a symptom of a deeper underlying pathology. The problem is, without question, a complicated one. Were this not true, the etiologic factor, and perhaps a mode of cure and a method of prevention, would have been developed long ago, for much clinical investigation and serious research has been performed, as well as no small amount of logical theorization and some sheer speculation. Though we have amassed much valuable information, we still lack many important facts, and there still remain quite a number of pertinent questions unanswered.

On the one side appears the clinical state of a patient with symptoms easily recognized as arising from a renal calculus, as perhaps he is suffering his primary renal colic from nature's effort to expel such a stone; surgery intervenes, and a crystalline concretion up to 1 cm. in diameter is removed. This stone is composed of crystallized salts normally present in the urine; it is not a foreign body; it is, and it has to be, the product of some morbid process.

Contraposed to this clinical state, and in an effort to explain it, stand today five pertinent theories as to why such a stone developed. Each theory is supported by interesting and acceptable research bearing on experimental stone production, with substantiating clinical studies suggesting a close relationship between the theory and fact. It is my purpose, first, to analyze briefly these five theories and to show the fallacies of each, or at least their shortcomings. It is only fair to state that in no research to date has the

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actual origin and the growth of a stone been traced from the first deposit of a tiny crystal of microscopic size, through the stages of added salt deposition, to macroscopic size when clinical symptoms may have supervened. We have accepted the theoretical inferences whole-heartedly, and with the simple-minded attitude so aptly expressed in the famous remark of Topsy, "I just growed."

THE THEORY OF STASIS.—The theory is simple: That when faulty drainage causes stagnation of the urinary flow, sedimentation and crystallization of urinary salts can take place about any nidus. The picture is familiar in vesical calculus as a complication of prostatic obstruction with residual urine, and one transfers this picture to the renal pelvis, where somewhat similar conditions of urinary stasis, both congenital or acquired, do occur.

Clinically, the incidence of hydronephrosis with stone is well known; with pyonephrosis stone is more frequent; and one is asked to include those possible states of faulty renal drainage that theoretically accompany the necessity of being long bedridden.

Experimentally, such hydronephrotic conditions have been created with some success in the production of calculi, while the introduction of foreign material, as infection, regularly increases the incidence of such experimental stone formation.

Discussion.—This theory—that stasis causes stone—though active in certain cases, fails utterly when such stasis can be proven to be absent. Also, it must not be overlooked that the finding of a stone in an hydronephrotic pelvis does not prove that the stasis caused the calculus; for often the reverse is true—that the stone came first and caused the hydronephrotic condition. Since the introduction of intravenous urography it appears that by far the larger percentage of renal calculi, especially when seen early, can be clearly proven to occur in kidneys totally devoid of stasis or faulty drainage, and in patients actively employed at normal occupations. Again, neither is it a constant fact, nor clinically is it expected, that every kidney pelvis which is poorly drained, even if infected, will sooner or later form stone; and the picture again is comparable to the occurrence of vesical calculus in prostatic disease, where, according to the highest figures published, calculus occurred in but 20 per cent. As germane to the subject at hand, let it be stated here that the material to be presented later especially excludes stasis as a factor in the etiology of stone, and in the clinical cases that will be shown only those proven to be free of stasis will be considered.

THE THEORY OF INFECTION.—In this theory both clinical studies and experimental work come closer to a solution of the problem.

Clinically, we have the rather prevalent finding of infection in operative cases (which unfortunately are seen late in the disease), until an unproven axiomatic statement has gained rather general credence, "that if every case of renal calculus be traced to its ultimate end, an infection would be found underlying it." Some highly suggestive observations have been made: The prevalence of calculi as a complication in chronic infections of bone; chil-

dren with Pott's disease; long-standing osteomyelitis cases; the observations during the late war in severe bone injuries with long drainage; and similar cases in industrial surgery; all pointing to a close relationship between chronic infectious processes, especially if such have periods of exacerbation, and the occurrence of renal calculi. Of real importance is the clinical observation of the rôle that infection plays in the recurrence of renal calculi and the incrustation of urinary fistulae, especially with that group of organisms capable of breaking down urea into ammonia, causing a persistent alkaline urine and the rapid precipitation of urinary phosphates.

Experimentally, it is quite essential to our clear understanding of the relationship between infection and the etiology of stone to realize at the very beginning that it is almost impossible to implant an infection in the undamaged and unobstructed urinary apparatus. Even in those cases found with stone and infection present, it is faulty reasoning to assume that the infection preceded the stone. It is a uniform observation, in both clinical and experimental work, that infection always increases the incidence of stone occurrence (or recurrence) and augments the rapidity of growth of calculi. The experimental work under this theory culminates in the report of Rosenow and Meisser, where they infected the devitalized teeth of dogs and inoculated the pulp cavity with cultures of "specific" Streptococci obtained from urinary calculi, and obtained 60 per cent positive results. This work has been neither corroborated nor challenged and disproved. It stands as probably the nearest approach to the clinical picture by the purely experimental production of renal stone; though it is to be stated clearly that these authors do not actually show us how, or where, or why, a calculus develops.

Discussion.—The theory of infection accepts such metastatic organisms as producing a pyelitic infection, and assumes that such bacteria, or their morbid products, create the nidus about which crystallization forms a stone. It does not admit that sterile cases of stone occur—which clinical studies unquestionably confirm—nor does it agree with the modern belief that organisms do not, and cannot, pass through the kidney without producing lesions. It is regrettable, on the other hand, that clinical cases too often are seen so late that secondary infection cannot be ruled out; but to anyone who cares to focus his attention only on the early cases—the small calculi, the primary attacks—it becomes increasingly evident that infection *per se* is not a part of the picture of either the clinical state of the patient, nor the laboratory studies of cultures of the affected renal pelvis. Moreover, the experimental workers have not traced the complete story from infection to actual stone formation, and have taken too much for granted between theory and fact. As will be presented later, our investigations contravert the rôle of local infection entirely.

THE THEORY OF VITAMIN DEFICIENCY.—This theory is beginning to crystallize, under its heading, the entire rôle of diet in stone formation.

Clinically, it attracts attention from two distinct angles: First, and perhaps of the greatest importance, is the realization that stone of the bladder in

childhood has almost disappeared from the picture of surgery. This change has occurred within the memory of living man, and without the medical profession taking any active part in advocating steps for its deliverance from this true scourge. The figures show that one hundred years ago in Europe and American over one-half of the operations for vesical stone were performed upon children. Today these operations are a rarity, and undoubtedly the liberalization of the dietary of infancy and childhood is the sole responsible factor. Second, is the realization that it is vitamin A, and to a lesser extent vitamin D deficiency, that is responsible, causing a disturbance of the calcium phosphate ratio. The clinical importance of this knowledge is rapidly growing, especially in the field of postoperative prevention of stone recurrence.

Experimentally, much could be told, since 1917, when Osborn and Mendel reported their observations in rats following certain diets. Actually, no tissue suffers more severely from want of vitamin A than the epithelium lining the urinary tract (McCarrison). Epithelial degeneration, desquamation and keratinization are characteristic of the condition. In experimental animals calculi do appear in the renal pelvis, and more frequently in the bladder, the frequency of which is in direct ratio to the length of time avitaminosis is continued. Other related factors are the consistent elevation of the p_H value in the urine to marked alkalinity, and the fact that the incidence of urinary infection is practically equal to that of stone formation.

Discussion of this theory must be confined to those factors known to be truly relevant. First, it is to be pointed out that the calculi that form under conditions of avitaminosis are consistently made of those salts known to have their normal ratios disturbed; that is, calcium-phosphate. Stones of uric acid, urates, or even calcium oxalate, do not participate in the picture at all. If the phosphorus is radically diminished in the diet, calcium carbonate can be made to crystallize, and occasionally calcium and magnesium phosphate have been observed. Second, is to be noted the alkaline urine and the high incidence of urinary infection in the experimental rats; factors not unusual in clinical urologic surgery, and in keeping with our knowledge of the ready precipitation of phosphatic incrustations under such conditions. Third, it appears that the earliest formation of crystalline material is about desquamated epithelial cells acting as a nidus, and vesical stone is much more frequent than renal stone, though neither appears until marked epithelial changes have developed. It is of interest to point out the possibility that large numbers of an apparently otherwise healthy population may be living under varying degrees of avitaminosis. The perfection of the Jean's test for deficiency in visual purple, as a delicate means of estimating slight degrees of vitamin A deficiency, is a most promising clinical aid; though at present this theory must limit itself to the calcium phosphate stone as met clinically.

The Theory of Colloidal Chemistry.—This fascinating supposition lacks only the two essentials of tangible fact and unquestioned truth. It assumes the recognized action of colloids to hold in solution the crystalloids of the urine and, not only to hold them in solution, but in a supersaturated state.

It is one of the many ingenious provisions of nature whereby it rids itself, even under physiologic conditions, of considerable amounts of excreted solid substances in the least possible quantity of fluid. The normal daily amount of colloid is sufficient for the elimination of the normal daily amount of crystalloid; but it is a delicate balance and is, therefore, assumed to be quite unstable, with a constant tendency to gain a more stable condition by having the crystalloids fall out of suspension. If one disturbs this so called colloidal balance by either increasing the crystalloid, or decreasing the colloid surface area, there occurs a precipitation of the crystalloids and their appearance in the urine as actual insoluble material. Such may be called the clinician's simple viewpoint of the intricacies of colloidal chemistry, in which one can hardly expect the physiologic chemist to wholly concur.

Clinically, it is pointed out that infection and epithelial degeneration are recognized disturbers of the colloid mass, either actually depreciating the amount, or causing coalescence with loss of surface area. Again into this theory will come all cases of hyperexcretory nature, where there is eliminated actually more crystalloid than there is colloid to hold it in solution.

Experimentally, this has been tested successfully by feeding animals excessive amounts of crystalline material, and so producing a condition of hyperexcretion. With the feeding of oxamid in large quantities, actual calcium oxalate stones have been produced, and recently Keyser (1936) has accomplished the same end by pushing calcium carbonate to the point of hyperexcretion. The remaining experimental work on the rôle of colloidal chemistry becomes so involved in theory, and so remote from clinical fact, that it bears little relation to the clinical problem at hand; though mention should be made of the suggested rôle of colloid material as the primary gel on which crystallization starts, and the colloid matrix to be found in the lamina of all calculi.

Discussion of this theory may be brief, for there is too little known of the intimate action of colloidal chemistry in both renal secretion and urinary excretion. Certain it is that fallacies creep in, for we all see patients with chronic phosphaturia, oxaluria and, best of all, cystinuria, who for years live with some such perverted metabolic balance and yet, even in this hyperexcretory state, fail to form stone. Moreover, this theory, though interesting in many ways, does not open any plausible lines of thought which may be subjected to experimental proof as actually causing a stone to originate. But there is to be said at this point that our knowledge regarding the colloidal balance of the urine, and the supersaturated condition in which urine is excreted, probably does play an important part in the determination of the type of salt to be deposited, once the causal factor is recognized.

THE THEORY OF PARATHYROID HYPERFUNCTION.—This latest addition to the theories bearing on the etiology of renal stone is probably the most positive of all.

Clinically, we now know how to recognize hyperparathyroidism, and in its recognition we have an explanation relative to the disturbance in calcium

metabolism and the characteristic bony changes in generalized osteitis fibrosa cystica. The metabolic disturbance is accompanied by a high tide of calcium in the blood and an increase of calcium in the urine. At the same time there is an increased urinary excretion of phosphorus in spite of a recognized decrease of serum phosphorus. Here we have, from a spontaneous clinical state, a typical reproduction of a hyperexcretory state under which stone has been made to form in experimental animals. This is not to be passed over lightly, for renal calculus has been recognized in 65.7 per cent of the 35 cases of hyperparathyroidism studied at the Massachusetts General Hospital.

Experimentally, the investigative work is being rapidly pushed, for the production and utilization of parathormone have given us a valuable aid in reproducing this pathologic state. There are no details to report as yet.

Discussion.—Valuable as this observation is, and as closely related to renal stone as it appears to be, there is lacking at present a definite relationship between parathyroid dysfunction and all urinary concretions. The disturbance is only in the calcium-phosphorus balance, and one wonders how it could play a part in oxalate, urate or uric acid calculi. In fact, the answer is again to be found in the studies at the Massachusetts General Hospital, for in a series of 300 cases of renal calculus they could prove hyperparathyroidism present in but 5 per cent.

That no one of these five theories fulfills the demands of clinical medicine and the recognized vagaries of the chemical composition of stone is the reason that each remains but a theoretical picture of the problem. Later there will be pointed out a possible relationship wherein the theory of parathyroid hyperfunction, as well as the theories on avitaminosis and that on colloidal imbalance, and perhaps that of infection, can be definitely interrelated, and perhaps found individually responsible, at times, for the causation of stone growth in the kidney. When one considers these five theories, sees their divergent angles, realizes their indisputable deficiencies and, at the same time, recognizes the varying chemistry of the stones themselves (where seven or more salts are involved and frequently present in pure or in mixed states), one feels that Howard Kelly was quite within the bounds of reason when he wrote: "No stretch of chemical or physiologic imagination will permit so heterogeneous a group of compounds to be ascribed to a common origin, or their deposition, in kidney, ureter or bladder to be uniformly charged to an identical cause."

It is the purpose of this study to trace the trail that has been followed during the past seven years, working through theory to certain facts that, of themselves, created an hypothesis. From this hypothesis two postulates were drawn and the work of proof inaugurated. There has been an effort to work back to what might be called a study of calculo-genesis; the stone's conception; its embryonic growth; its fetal life cycle; up to the point where size and symptoms made it a clinical entity and clothed it with raiment familiar to surgeons.

With this background of the common knowledge on the etiology of renal calculus, we wish to point out, very briefly, the various experimental research problems that have absorbed our interest and endeavor, omitting many details for the sake of brevity, but outlining the train of reasoning that has been followed.

(1) An analysis of calculi was made to determine the chemical characteristics, thinking that perhaps the problem had to be broken down into its component parts in order to determine the individual origins. It was assumed that perhaps the cause for the growth of an uric acid calculus was entirely different from that of a calcium oxalate stone, and that perhaps a different chapter would have to be written for each variety of chemical composition, with a differing etiology for each. This problem has not been productive of results. Three hundred and ten calculi have been analyzed* and compared to the history and clinical studies without finding any specific lead between cause and effect. In this series calculi composed of uric acid, sodium urate, ammonium urate, calcium phosphate, calcium oxalate, calcium carbonate, calcium-magnesium phosphate, calcium-magnesium and ammonium phosphate, and cystine have been encountered. These studies in part have been published.

(2) We tried to infect the renal pelves of dogs by implanting organisms by cystoscope and ureteral catheter. Several exogenous and endogenous organisms were used, and when results were not forthcoming, organisms cultured from stone cases, both renal pelvis cultures and cultures from the center of calculi, were tried, but again without success.

(3) Realizing the improbability of being able to implant a culture in the healthy renal pelvis of the dog, we repeated some of these attempts, and endeavored to traumatize the renal pelvis by a wire projecting from the end of the ureteral catheter. Again success was so uncertain that the problem was abandoned.

(4) The foregoing failures taught us that a lesion was necessary to effect the implantation of an infecting agent. Twelve dogs and eight rabbits were employed. In some a pyelotomy was performed and different kinds of trauma to the pelvic walls effected by instrument, fulgurating electrode, or chemical burn. These pelves were then infected with organisms from a man who had had seven operations for recurrent urinary calculi. In others of these animals one or both ureters were opened in the middle third, and the traumatizing instrument passed to the pelvis under hand guidance, in order to keep the operative field at a distance. In this series we effected six lesions—five

* Of the kidney calculi, numbering 151 cases, 71 of the stones, or 47 per cent, occurred as pure salts, as follows: Calcium-magnesium phosphate, 39; calcium oxalate, 11; triple phosphate, 7; uric acid, 6; calcium carbonate, 4; cystine, 4. Of the ureteral calculi, numbering 159 cases, 100 of the stones, or 62.9 per cent, occurred as pure salts, as follows: Calcium-magnesium phosphate, 49; calcium oxalate, 39; uric acid, 7; calcium carbonate, 4; cystine, 1. The remaining 139 cases had stone or stones of mixed salts in 28 differing combinations. I am deeply indebted to Drs. H. G. Beeson, C. A. W. Uhle and P. D. Melvin for this painstaking routine study.

successful implantations of infecting organisms—and in two dogs we were able to produce visible sand or gravel. In the eight rabbits we produced six lesions, of which three held the infection, two healed, and one unhealed was sterile at autopsy. Triple phosphate sand was found in one.

(5) A group of rats were put on vitamin A deficiency diets, and a search for characteristic lesions was made. These rats were part of an extensive vitamin A problem. In some we obtained evidences of early aseptic ulcerations, but the vitamin deprivation was marked, and death occurred too early for the characteristic calcium phosphate deposits to be formed. This series is now being repeated with 96 rats at the Wistar Institute as part of a further study of the problem.

(6) During this period of experimentation clinical cases were watched, searched and studied to correlate chronic focal infections with the occurrence of renal stone, and it may be noted in passing that these studies do constantly bear evidence of such close relationship, and deductions therefrom have been published. But in this research we were unable to obtain other than the suggestive finding between theory and fact exemplified in the experimental problems or the clinical studies.

Such were some of the lines of endeavor that were pursued in the hope of finding leads which would be promising to follow in order to experimentally produce stone and allow of clinical interpretation or further study of the early changes that are creative of calculus growth. It is perhaps now evident that our entire line of thought was predicated upon the firm belief that renal stone is but a symptom, developing upon some underlying and preexisting renal pathology; and most of our efforts were bent toward making an infection the basic and fundamental primary cause.

If one divorces oneself from the plethora of theory in this field, and looks at the problem from a more detached point of view, certain questions arise that are extremely helpful to a clearer insight into the problem as a whole.

One cannot dwell upon the origin of primary renal calculus without, sooner or later, forming a firm conviction that there has to be an initiating lesion. Everything points toward it, every known fact strongly suggests it, every pertinent question demands it, and pathology itself is incomplete without it! Stone is a symptom, and not a disease entity. Stone is made from the common salts in the urine. These salts exist in a supersaturated state. Stone will grow on any foreign tissue or foreign body. Stone has got to be a gradual accretion of crystals demanding a nidus for the seeding of such crystallization. Stone requires time to grow and, therefore, must be stationary and fixed in its beginning in order to gain clinical size. Therefore, as our researches progressed, though they were unproductive of positive results, they at least seemed to help to crystallize in our minds the idea that all renal calculi must have, as a point of origin, some preexisting lesion in the renal pelvis. It was the growth of this idea—the absolute necessity of such an initiating lesion—that led us on and that suggested the above experiments with trauma and infection.

Gradually in the course of our researches there was evolved a hypothesis which, as stated, predicated the origin of any stone upon the existence of a previous lesion, and we made as our first postulate: *That there must be an initiating lesion that precedes the formation of a renal calculus.* It was perfectly evident that in none of the researches on renal calculus had any one observed the actual origin, development and growth of a stone. Nowhere could be found the stages between theory and fact. Even those who had succeeded in producing calculi experimentally had not attempted to trace the actual onset and developmental stages, and practically all left one with the impression that a stone floated about in the renal pelvis while it grew in size, or else some perversion of nature nurtured this aggregation of crystals until it was at least large enough to produce a renal colic. The story of what might be called "calculo-genesis" had not yet been written.

If our first postulate demanded an initiating lesion, it naturally became evident that a second postulate would be necessary as to where such a lesion might be expected to be found. It was not difficult to exclude the probability that the simple lining epithelium of the pelvis and calices would produce the initiating lesion; their resistance is high and their anatomy and physiology are simple. The renal papilla on the other hand is quite the reverse; performing a complicated function, open to multiple physiologic variations, and known to suffer recognized insults that lead to pathologic changes. Therefore, a second postulate was formulated: *That the initiating lesion was to be looked for on the renal papilla.*

A search of the literature for what is known of the pathology of the renal papilla was most disappointing. The hemorrhagic papillitis of Fenwick, the uric acid infarcts of the newborn, the calcium infarcts of Henle, and the occurrence of tuberculous ulcerations (Lieberthal) cover about all that have been recognized as primary lesions. As our experimental efforts had been abortive of results, it was decided to go to the autopsy table and, by dissecting out each minor calyx to the complete exposure of its individual papilla, to uncover whatever observations awaited us, and to hope that time, patience and industry would reward our efforts by possibly showing us lesions that could be related to stone's origin and growth and, perhaps, to the finding of early crystalline deposits. In other words, instead of trying to *create* stone by following theoretic dogma, we turned to trying to *find* stone as it occurred in man, and to observe it in its incipiency, its point of origin and its causal factors. The cooperation of the Department of Pathology at both the Hospital of the University of Pennsylvania and the Abington Memorial Hospital made this effort possible and, at my request, each put at my disposal the kidneys from all autopsies, to be opened and sectioned as we saw fit. These kidneys were examined by first opening the pelvis, and from it, by careful dissection, each major and minor calyx was laid open in turn and each papilla completely visualized and studied with a

hand lens. During the past six months the autopsy material from the Philadelphia General Hospital has been added, though here the kidneys have been routinely examined before we receive them.

Starting in December, 1935, there have been examined to date (February 15, 1937) 429 pairs of kidneys. Of this total number 73, or 17 per cent, have shown papillary lesions, either unilateral or bilateral, of one or more of the renal papillae. As above stated, our interest has centered particularly about what might be called the minor pathologic changes, and kidneys showing gross destructive pathology—pyonephrosis, advanced tuberculosis, etc.—are not included at all. In other words, kidneys heretofore passed as having normal pelves have been the ones we particularly searched for early papillary lesions.

Let me digress a moment to draw attention to two clinical observations that have been repeatedly noted by us, and which should be mentioned at this point as bearing upon this research and to a degree suggesting it. First, the small calculi, which we have studied over a number of years, consistently showed a certain peculiarity; especially those whose clinical history was of short duration, evidencing that there had been but a brief interval from the time of becoming clinically obstructive to the moment when they were removed from the urinary tract. Hand lens examination of such calculi regularly showed a surface highly crystalline, but also a portion which was smooth and somewhat depressed, resembling a facet. This facet was so consistent in its occurrence in these clinical cases that it was early taken as suggestive evidence that the calculus had had a mural attachment. Such small calculi have been photographed and enlarged, have been sectioned and polished, and the observation has been repeated so many times that it stands as quite positive circumstantial evidence of the mural origin and attachment of stone while attaining what might be called clinical size. Secondly, this suggestive observation has made us study our roentgenograms more closely for the localization of small stone shadows, and since the introduction of intravenous urography, we have been pleased to find that such early calculus shadows can be proven to occur most regularly in the minor calices.

Starting our postmortem series we examined 27 normals before encountering our first papillary lesion. This lesion was so innocent in appearance, and yet so definitely abnormal to our previous observations, that it demanded attention. There was present a cream-colored area near the tip of the papilla, which was definitely not on the surface, but appeared to be sub-epithelial. Shortly thereafter we found four kidneys that showed a similar lesion—some with a solitary plaque on only one papilla, others with several papillae involved, some unilateral in occurrence, others showing bilateral papillary involvement. On microscopic study, the lesion was found to be a plaque of calcium deposited in the interstitial tissue of the renal papilla, and definitely not intratubular. Special stains were used to identify this as calcium, and

material has been collected by teasing out such plaques, which on being analyzed chemically was proven to be calcium.*

It is my wish at this point to especially emphasize that this lesion, as we have found it, occurs particularly in the walls of the renal papilla—some centrally placed, some just subsurface—that it is not an intratubular deposit, but a broad plaque of calcium invading and replacing interstitial tissue (Figs. 1 and 2); that, from our studies, its first deposition is in the collagenous, or basement membrane of the terminal tubule walls, from which it gradually involves the intertubular spaces and causes the tubules to gradually shrink, lose their lining cells and disappear, or remain with markedly narrowed lumens; that in the many sections which we now have studied, of practically all the lesions above mentioned, in only two has there appeared cellular destruction and round cell infiltration, which could be interpreted as evidence that infection accompanied this calcium deposition. These exceptions I consider as the inevitable finding when taking such material from the autopsy table, and consider that it represents a secondary factor where infection has been implanted upon a preexisting pathologic condition.

It was not long before a kidney was found where, on such a calcium plaque, a secondary deposit of some black material could be seen. This deposit was about the size of the dot over the printed letter "i," and quite as distinctly visible (Figs. 3 and 4). Sections of this specimen showed evidence that, through increasing growth and pressure, this calcium plague had lost its epithelial covering, and on its surface was deposited a layer of some different material. Its minute size defied analysis and specific staining methods; but from our subsequent studies we now look back upon this tiny speck as the earliest evidence of renal calculus formation ever seen.

Our next important step arrived with the examination of a kidney in which a small stone was found, which was, however, unfortunately dislodged by the knife as the kidney was opened; search, however, revealed another papilla which contained a visible calcium plague, upon and from which a second stone projected into the lumen of the minor calyx (Figs. 5 and 6). This stone was firmly adherent, measured approximately 2 Mm. in diameter,

* Dr. James H. Jones of the Department of Physiologic Chemistry of the University of Pennsylvania analyzed 4.65 mg. of such material for me, to find calcium to occur in 4.967 per cent (5 per cent). As muscle, liver, lung and kidney contain only 0.1 to 0.5 per cent, it left but little doubt that calcium was present in high concentration. Dr. Charles G. Grosscup of the Abington Memorial Hospital, with 1.9 mg. of material, similarly composed of plaques teased from visible papillary lesions, undertook a quantitative analysis. He reports that the material was microcrystalline and chalky in appearance, was insoluble in water and acids, but dissolved in alkalies. Quantitatively calcium was present in 19 per cent, nitrogen in 10 per cent, CO_2 in 1 per cent, and phosphorous present but difficult to make accurate quantitative estimation. He states, "In view of the low CO_2 and phosphorus content and high calcium (19 per cent), it was evident that about 80 per cent of the calcium must be bound in some other form. With the murexide test indicating xanthine, the insolubility of the material in acids and the high nonprotein nitrogen suggest that this may be nucleic acid."

and resembled in appearance the larger calculus which the knife had dislodged. Study of these specimens has been most valuable: in the first place the dislodged stone has been analyzed and proven to be composed of calcium phosphate; microscopic sections of the smaller, adherent stone have demonstrated unquestionably that it is growing from, and is supported by, a typical calcium plaque imbedded in the wall of the renal papilla; special stains have shown that the stone is of calcium phosphate, while the plaque itself is composed of calcium but does not show phosphate as a salt. So here we have a definite renal calculus growing on an initiating lesion, and the lesion is a deposition of calcium in the walls of the renal papilla, and the two are of different chemical composition. The demonstration of this fact becomes a most vital factor in our conception of the origin of renal calculus, and should be emphasized. Here, for the first time, we find a lesion of not infrequent occurrence (17 per cent), and of rather constant character and chemical composition. This lesion, innocent enough while buried in the wall of a renal papilla, can lose its epithelial covering and from then on be bathed in calyceal urine, and acting as a foreign body, it becomes the nidus upon which urinary salts precipitate. Here, likewise, we can picture the reason how and why renal calculi can remain stationary while increasing in size. Here we can account for a common origin of stone formation, which also allows of the known variation of salts so deposited to form stone, and one can assume that the salt which does crystallize to form a calculus is the one which, at that epoch, is most readily thrown out of suspension. Study of the serial sections through this stone has likewise shown us, at the edge of the plaque, a definite elevation of the plaque by the encroachment of crystallization, which suggests that when a stone becomes free, it does so by tearing the plaque from its tissue bed and taking it away with it.

From this point on our studies have taken a variety of directions. We have observed to date 14 kidney specimens, in which calculi have been seen growing upon papillae. Eight of these have had a single stone present, in five of them two papillae were found with stones adherent, and in one case all six papillae supported stone deposits. This makes a total of 24 observations where calculi have been found actually adherent to, and growing upon, a renal papilla. To these may be added four other specimens where the calculus was less than 1 Mm. in diameter, but which were as plainly visible

FIG. 1.—Drawing of a typical subsurface calcium plaque in the wall of a renal papilla.

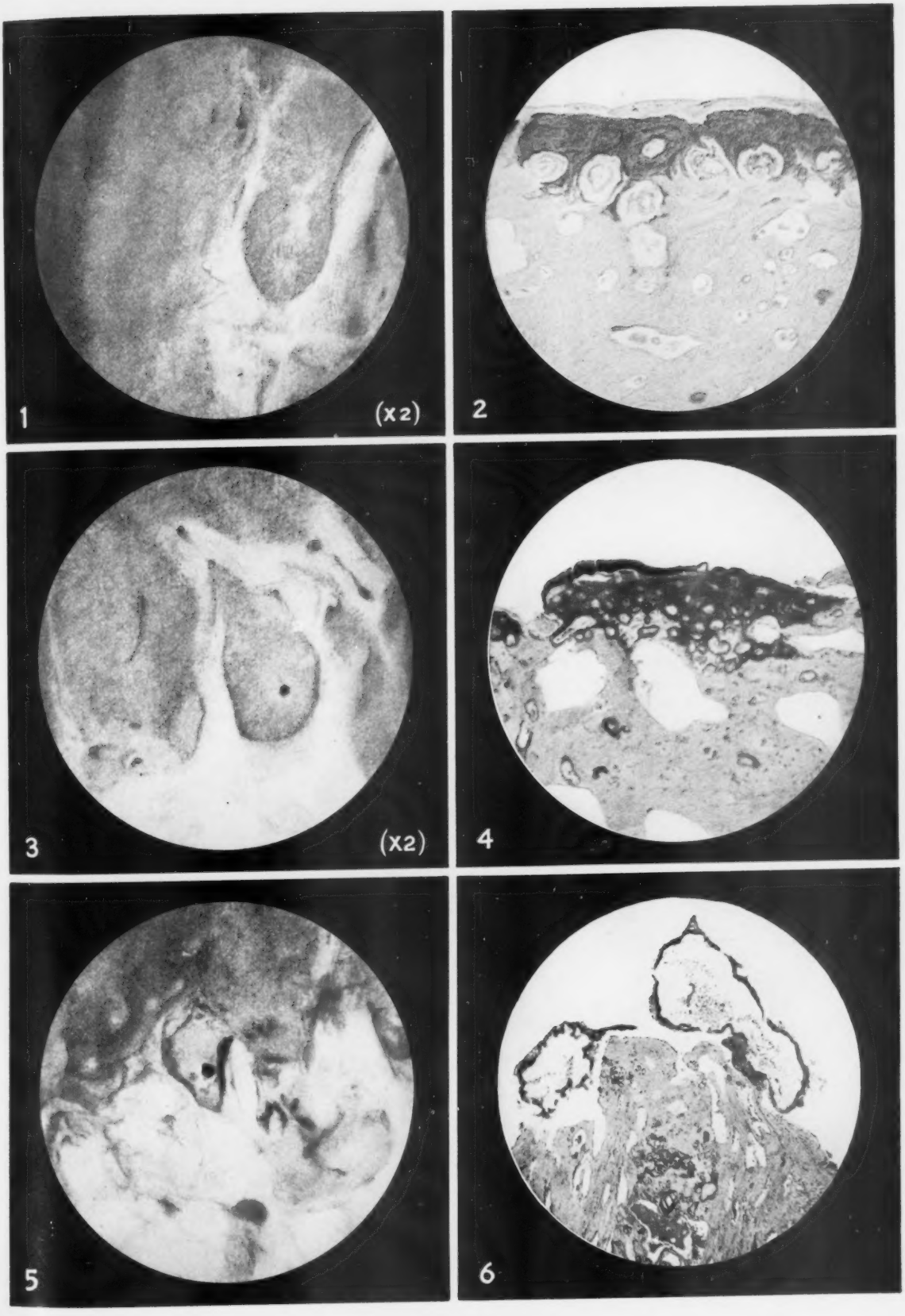
FIG. 2.—Detailed drawing of high magnification of a subsurface calcium deposit on the renal papilla. Note the shrunken tubules at the base of the plaque, their loss of normal epithelium, and the absence of any reaction suggesting infection.

FIG. 3.—Colored photograph of a renal papilla, showing the subsurface calcium deposit, and in its center a tiny black secondary deposit. This the earliest evidence of secondary deposit which forms stone.

FIG. 4.—Colored photomicrograph of Fig. 3, showing the calcium plaque which has lost its covering mucosa, and on which is a secondary deposit of brown material, taken to be the earliest evidence of renal calculus formation. No evidence of infection.

FIG. 5.—Colored photograph showing calcium deposits in two papillae, and a stone attached and growing on the calcium deposit in the third papilla. Another stone of similar character found in this kidney has been analyzed and proven to be composed of calcium phosphate.

FIG. 6.—Colored photomicrograph of Fig. 5, showing the papillary stone attached to its calcium plaque. The differential staining shows the stone composed of calcium phosphate. The plaque with some phosphate staining; counterstains for calcium of different composition. No evidence of infection.



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as a cinder on the cornea of the eye. Each of these has been sectioned and this earliest deposit proven microscopically.

It has been our purpose to try to demonstrate that each of these calculi was supported by a primary intrapapillary calcium plaque as its initiating lesion. That this is exceedingly difficult will be readily understood. In the first place such calculi have to be properly decalcified before sectioning—not completely decalcified, of course, but enough to allow sectioning—and in doing this we have lost specimens. Again, we have tried to depend upon special stains in an effort to differentiate the various possible salts, which has required special methods of tissue fixation. This, in turn, has dissolved some specimens, and others have been made so brittle as to be completely fragmented on sectioning; and this has resulted in the loss of some specimens. In addition, it has been difficult, and most tedious, to attempt complete serial sectioning of stones 6 and 8 Mm. in diameter, in order to cut through the stone and its possible underlying plaque, which frequently is minute and generally eccentrically placed. In order to avoid these disappointments, two other procedures have been adopted in order to prove that each calculus arises from a calcium plaque in the wall of a papilla. First, a few selected specimens have been subjected to tissue-clearing methods, and by this means we hoped to visualize through the cleared papillary wall the underpinning of the calculus upon the papilla's surface. The second method was discovered accidentally, when a tiny black stone, 3 Mm. in diameter, was inadvertently dislodged; on examining it under a strong lens, it was seen to be distinctly composed of a black mass with a bossed surface, simulating the early jackstone development of the calcium oxalate stone, but to one surface a clear-cut white layer was adherent, distinctly different and definitely crystalline: this we inferred to be the calcium plaque. Through the cooperation of Dr. A. Newton Richards these two layers were analyzed separately. The calculus weighed 5.5 mg. (Fig. 7). Microchemical analysis was made, which showed that the white layer, or plaque, was composed of calcium carbonate, with a decided trace of calcium phosphate. The black stone gave a negative test for calcium carbonate, calcium phosphate and uric acid, but was conclusively proven to be pure calcium oxalate. This is a most significant finding, for here again, on a papillary plaque composed of calcium carbonate and calcium phosphate, a stone composed of a different salt has formed (calcium oxalate). Two other calculi of similar character have been gently dislodged from their papillary attachments, and in each case the white plaque is plainly visible. This, to a degree, supports the assumption that as a calculus is extruded in life, it gains its freedom by tearing away its supporting plaque from its original tissue bed.

While these researches have been furthered in an effort to substantiate the idea of a papillary initiating lesion, and to quite definitely prove the rôle played by the finding of the calcium plaque formation and its relationship to the clinical occurrence and growth of a renal calculus, we have been keenly interested also in trying to solve the raison d'être of the deposition of cal-

cium in the papillary wall. This work is far from finished, but it is possible, at the present time, to give the pathology as we have interpreted it. I am obliged especially to Dr. John Eiman, Pathologist to the Abington Memorial Hospital, for the following detailed description and to Dr. Balduin Lucke, Professor of Pathology, University of Pennsylvania, for his interest and aid in obtaining special staining methods and in section cutting, as well as his coincidence with the opinions expressed on the microscopic pathology.

Microscopic Pathology.—*Normal Papilla:* The normal papilla is covered by cells which differ from those lining the calices and the collecting tubules, in that they are a single layer of flat, very thin cells which are modified cells of the lining epithelium of the collecting tubule. The collecting tubules in the papilla vary considerably in diameter, the larger ones, or the ducts of Bellini in the area cribosa on the apex of the papilla, measuring from 100 to 200 microns in diameter. The cells lining the collecting tubules are arranged as a regular single layer, with their nuclei at one level and their free surfaces bulging slightly into the lumina. The smaller collecting tubules are lined by sharply defined cuboidal cells. As the collecting tubules grow larger, the cells become higher and in the ducts of Bellini acquire a tall columnar form. The epithelium of the collecting tubules rests upon a well developed and distinct basement membrane. The interstitial connective tissue in the papilla is more abundant than in the cortex and medulla of the kidney, and there is an abundant amorphous ground substance.

The Calcium Plaque: Figs. 1 and 2 (Specimen No. 36-7, U. of P.). Approximately midway between the tip and the base of the papilla, underneath the epithelial covering, is an area, measuring 2.5 Mm. in length and varying from 0.1–0.3 Mm. in width, composed of dense connective tissue. The surface of the papilla over this area is somewhat irregular. The blood vessels in this location are less numerous, and those present are partially obliterated by the surrounding dense connective tissue. Some of the collecting tubules are devoid of lining epithelium, while other tubules show cells evidencing degenerative changes. In the portion of this region nearest the tip there are a number of small areas showing granular detritus. In this location are seen two small irregular deposits of purplish-staining material (H. and E.) measuring approximately 0.3 by 0.03 Mm. By special staining methods* these deposits proved to be calcium. These calcium deposits, or plaques, are separated from the surface of the papilla by a thin irregular layer of connective tissue and covering epithelium. Where the calcium is less dense it occurs in rings, definitely deposited in the basement membrane and spreading into the ground substance of the surrounding connective tissue.

Throughout the papilla, but especially near the tip, there are areas showing an increase of interstitial connective tissue. Some of the blood vessels show narrowing and irregularity of their lumina. The cortex and medulla show no noteworthy lesions. The walls of the convoluted tubules show no calcium deposits. The larger vessels show a moderate degree of arteriosclerosis. There is no microscopic evidence of infection in this specimen.

(Specimen No. 36-42, U. of P.): Near the tip of the papilla is a calcium deposit measuring approximately 0.3 by 0.3 Mm. It is separated from the surface by a layer of connective tissue and covering epithelium varying in thickness from a few strands to .06 Mm. The surface of the papilla shows a practically normal covering except at one point near the plaque where there is a depression about .05 Mm. deep. Most of the collecting tubules near the plaque are devoid of their epithelial lining, but an occasional tubule shows small clumps of partly degenerated and desquamated epithelial cells. Blood vessels near the calcium plaque are diminished in number; they are distorted and show various degrees

*The special stains used in this study are: von Kossa, Eros, Giemsa, Masson, azocarmine, elastic tissue stain, Grandis and Mainini, methylene blue, and picric acid stain for urates and uric acid.

of compression to the point of obliteration. Around the dense calcium plaque are seen ring-like deposits of calcium in the basement membrane of the tubules. There is no microscopic evidence of infection in this specimen.

Fully Developed Initiating Lesion: Figs. 3 and 4 (Specimen No. 36-43, U. of P.). Near the tip of the papilla there is a loss of continuity of the surface with the formation of a shallow depression. In this depression is situated an irregular plaque, measuring 0.6 Mm. in length and from 0.1–0.2 Mm. in width, which by special stains was proven to be calcium. The plaque shows numerous small, roughly rounded spaces which vary in size. Over the surface there is an irregular layer of brownish material which measures 0.025 Mm. in thickness; this layer is definitely of different material than the calcium plaque. The calcium plaque is not uniform in texture; in the looser parts about the periphery the calcium is seen deposited in the basement membrane of partly compressed collecting tubules. There are a few tubules which show deposition of calcium in some of the lining cells, while other cells are completely preserved.

Special stains show that when the calcium is to be deposited, it appears to be laid down first of all as fine granules in the basement membranes of the collecting tubules. With further deposition such granules coalesce until the calcium appears as a complete ring encircling the tubule, generally with loss of epithelial lining, though occasionally there can be found some viable cells. From this point, calcium is further deposited in the ground substance of the surrounding connective tissue, with gradual generalized coalescence and plaque formation. There is no evidence of a deposition of calcium in the epithelium of the convoluted tubules.

Near the plaque and at the tip of the papilla are seen many large, roughly rounded, irregular spaces, many of them devoid of epithelial lining, but some showing epithelial cells in different stages of degeneration. These large spaces undoubtedly are cross sections of large collecting tubules or ducts of Bellini that have lost their normal epithelial lining. Toward the base of the papilla the dilatation of the collecting tubules is less marked, and the lining shows consistently larger numbers of epithelial cells of lesser degrees of damage. There is no microscopic evidence of infection in the specimen.

This we have termed the fully developed initiating lesion, for here we see, for the first time, a secondary deposit of entirely different staining properties laid down in laminae upon a simple calcium plaque: The essential point being that this plaque has lost its connective tissue and epithelial covering.

Calculus on Plaque: Figs. 5 and 6 (Abington No. 1). Near the tip of the papilla, 0.3 Mm. beneath the surface, there are irregular calcium deposits over an area 2.2 by 0.6 Mm. Near these deposits, on the surface of the papilla, there is an irregular calcium plaque measuring approximately 0.4 by 0.1 Mm. To the outer surface of this calcium plaque, and only to the plaque, is attached an irregularly shaped calculus measuring 1.75 by 0.8 Mm. This stone, by special staining methods, was proven to be composed of calcium phosphate, while the plaque on which it grows stains for calcium but not for calcium phosphate.

Here is microscopic evidence of a calculus of one chemical composition growing from the surface of an intrapapillary plaque of a different chemical composition.

All Lesions on One Papilla. (Specimen No. 32-682, U. of P.). This papilla shows a number of subsurface lesions. Near the surface on the side of the papilla there is a small roughly oval area, homogeneous in appearance, composed of dense connective tissue and showing very few partly degenerated nuclei. Close by, in a similar subsurface location, is a small necrotic area separated from the surface by connective tissue and covering epithelium. A short distance from this necrotic area toward the tip of the

papilla is another area of necrosis showing deposition of calcium, yet still separated from the surface of the papilla by three or four strands of connective tissue cells. Still further toward the tip of the papilla is seen a small depressed area devoid of normal covering, with irregular fragments of calcium deposit at its base. Apparently something has been torn away from this area, carrying with it part of the calcium plaque. In no place is there any evidence of infection, or of calcium deposits in the convoluted tubules.

This section pictures a most fortunate find, for from this papilla we removed a black stone and on it have demonstrated its attached plaque (Fig. 8). Also, as described above, are demonstrable in this section: (a) An undisturbed subsurface calcium plaque; (b) an area of necrosis as yet without calcium deposition; and (c) the earliest changes of simple fibrosis with some pyknotic nuclei (Fig. 9).

Discussion.—The general impressions obtained by a close study of these specimens are: That there occurs a definite damage to the epithelial lining

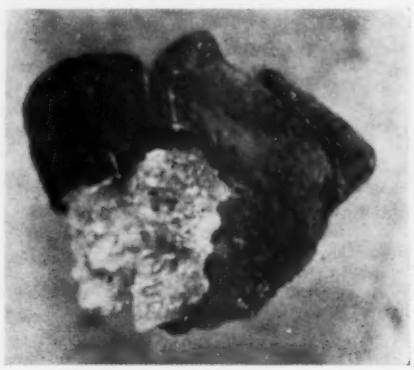

Fig. 7.—Calculus found attached to a renal papilla, which was manually removed. The specimen weighed 5.5 mg. and measured 3 Mm. across. The black stone has been proven to be composed of calcium oxalate: the white portion is the torn-out plaque from which the stone developed, and is composed of calcium carbonate and phosphate.

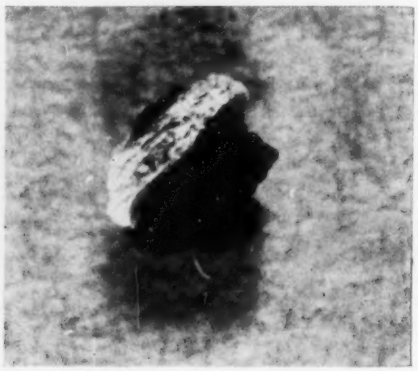

Fig. 8.—Tiny renal calculus removed from a renal papilla (Spec. 32-682). The calculus weighed approximately 1½ mg. and measured 1 Mm. This greatly enlarged photograph shows the black calculus and its firm attachment to the white calculus plaque, which was torn out of its tissue bed on removal of stone.

of the collecting tubules, and that the nearer one goes toward the tip of the papilla the more noticeable the changes are; that there is a marked damage to the ground substance of the interstitial connective tissue and of the basement membrane of many of the collecting tubules. Here and there the ground substance is broken up and granular, and has a necrotic appearance. These changes appear to be followed by the deposition of calcium and calcium plaque formation. This study also reveals that in places the calcium is deposited in damaged epithelium of the collecting tubules, but the primary deposit appears to be in the basement membranes leading to the formation of ring-like structures. No evidence of infection is seen in any of the sections presented.

Mention should be made of five specimens in which this simple pathologic process shows additional features. In two of these five specimens the presence of bacteria has been demonstrated. Whether these are secondary in-

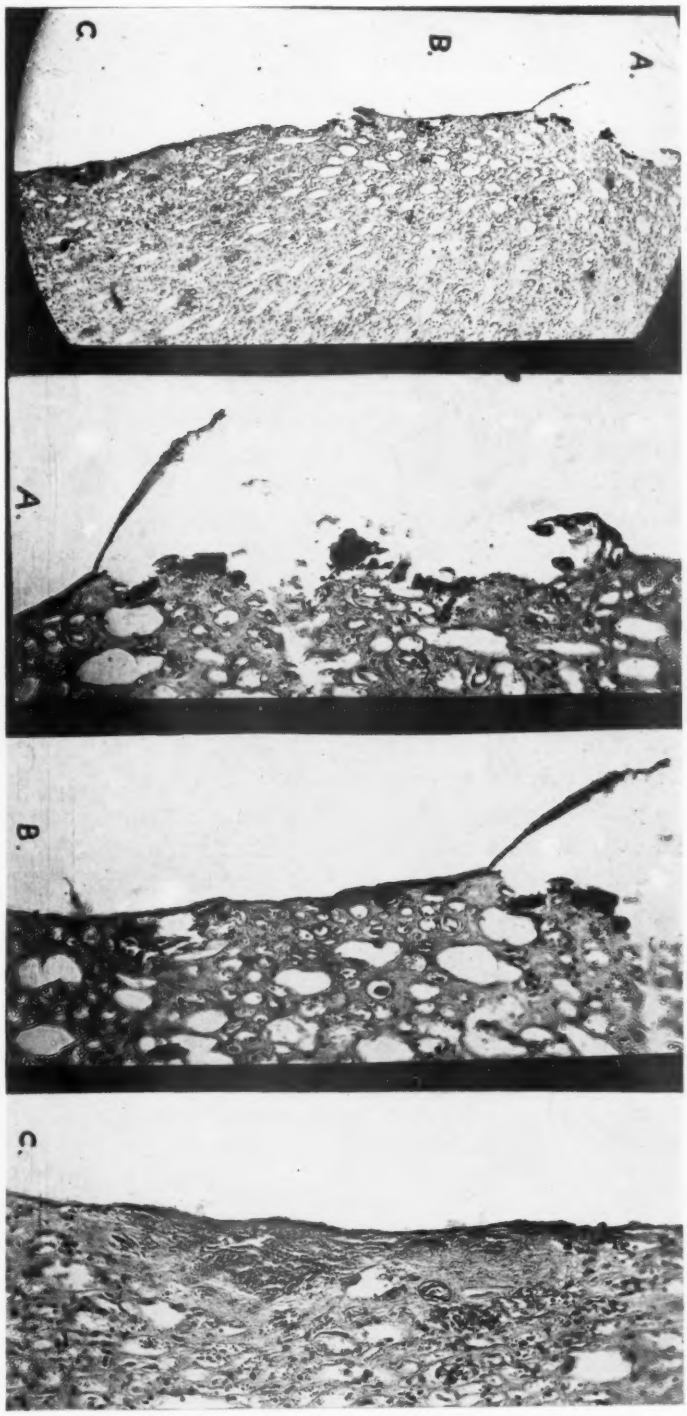

FIG. 9.—All lesions on one papilla. On the left is a photomicrograph (low power) showing at A an area from which the calculus shown in Fig. 8 was removed. At B is a subsurface calcium deposit; while at C is an area of ischemia and necrosis. Succeeding pictures are these three areas under higher magnification.

vaders, or play a more important rôle in the papillary pathology, we are not prepared to state at present. It is certain, however, that the tissue changes and the pathologic processes seem to be fundamentally the same, and the entire picture impresses one as a more rapidly progressing lesion, a more generalized involvement of the papilla, and it is to be noted that each of these patients died from a septic condition. All five of these specimens show multiple papillary involvement, with yellowish to brownish streaks running in converging lines toward the tip of the papilla. Microscopically are to be seen marked tubular damage and massive deposits of calcium phosphates and some urates. We recognize in this a picture similar to that originally described by Henle and thought by him to be (tubular) calcium infarction; a view later modified by Lubarsch who showed that the calcium deposition was also extratubular. Neither of these authors in any way attributed his observations to be related to renal calculus formation; but it is to be recorded that one of our cases in this group had two small renal calculi, composed of calcium phosphate, attached to separate papillae. We concluded, therefore, that though this group represents a rarer form of papillary pathology, it nevertheless is essentially the same process and is related to the origin of renal calculus.

With this interpretation of the pathology as unfolded on microscopic study, our researches naturally led to an effort to determine the basic cause for such degenerative changes. We are not ready, at present, to draw any conclusions from these studies, but the picture would not be complete unless mention were made of our ideas and the direction of our efforts. First, by reverting to the five theories relative to stone origin, we have undertaken to try to produce hyperparathyroidism by the giving of parathormone, and a group of dogs are undergoing such administration, to be sacrificed at selected intervals and their renal papillae studied. Secondly, a large group of rats are on vitamin A deficient diets, who are to be sacrificed at earlier intervals than in other studies, in order to determine the possible occurrence of early lesions. Thirdly, the rôle of infection *per se* is to be studied, both as to specific organisms and in further tissue studies which we are watching, but we are particularly interested in certain phases of toxin action as divorced from actual tissue infection. It will be of the greatest interest to see which, if any or perhaps all, of these previously suspected etiologic factors play a part in causing the initiating lesion which we are describing.

CONCLUSIONS

(1) An hypothesis has been formulated that renal calculus formation is dependent upon a preexisting renal lesion. Our studies conclusively point out that this is true, and that calculo-genesis is but a symptom of some form of renal papillary damage antedating the deposition of crystalline urinary salts.

(2) This hypothesis placed the expected lesion upon the renal papilla.

Microscopic studies have shown a hitherto unrecognized papillary lesion, consisting of the deposition of calcium in the walls and intertubular spaces of the renal papilla, which lesion has been observed in 17 per cent of the 429 pairs of kidneys studied at autopsy.

(3) To date, 28 specimens of renal calculus formation have been observed as growing upon, and actually adherent to, the renal papilla.

(4) In seven specimens we have been able to prove conclusively that the calculus was intimately attached to this calcium deposit, and to show that once the calcium deposit lost its epithelial covering it was subjected to a constant bath of calyceal urine, from which it is naturally assumed that crystals were deposited as upon any foreign body.

(5) We report the positive evidence that cases of calcium phosphate stone and of calcium oxalate stone have been proven to grow from such calcium plaque formation, and that in each of these cases the salt deposited as a calculus was of different chemical character from the plaque to which it was attached.

(6) It is shown that such calcium plaques appear to be a natural reparative process to some form of tubule damage, the occurrence of which is in much higher incidence than the actual clinical frequency of renal stone.

(7) The most significant conclusion to be drawn from this research is that the answer to the secret which has shrouded in mystery the true etiology of primary renal calculus is to be sought in an increasing knowledge of the physiology and pathology of the renal papilla.

ORGANIZATION OF THE AMERICAN BOARD OF SURGERY COMPLETED

In answer to the widespread demand for an agency which will attempt to certify competent surgeons, the American Board of Surgery has recently been organized. This Board is a member of the Advisory Board of Medical Specialties which includes all of the boards of certification for the different medical specialties which have been already organized. Since boards were in existence for the certification of practitioners of some of the surgical specialties such as ophthalmology, otolaryngology, obstetrics and gynecology, genito-urinary surgery and orthopedic surgery, it is expected that the American Board of Surgery will be responsible for the cerification of general surgeons as well as those practicing in the remaining specialized subdivisions of surgery.

Acting upon the invitation of the American Surgical Association, the following surgical societies cooperated in the creation of the American Board of Surgery: the American Surgical Association, the Surgical Section of the American Medical Association, the American College of Surgeons, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association, and the New England Surgical Society. The first three of these bodies which are national in scope have three representatives on the Board. All of the other societies have one representative each. The representatives of the cooperating societies are nominated by the society which they represent and upon approval of the Board shall become members of it. The term of membership on the Board will be six years. The following were chosen to represent the cooperating surgical societies:

Dr. Evarts A. Graham	Representing the American Surgical Association.
Dr. Arthur W. Elting	
Dr. Allen O. Whipple	
Dr. Donald Guthrie	Representing the American College of Surgeons.
Dr. Erwin R. Schmidt	
Dr. Harvey B. Stone	
Dr. Fred W. Rankin	Representing the Surgical Section of the A. M. A.
Dr. Howard M. Clute	
Dr. J. Stewart Rodman	
Dr. Philemon E. Truesdale	Representing the New England Surgical Society.
Dr. Thomas Orr	Representing the Western Surgical Association.
Dr. Robert Payne	Representing the Southern Surgical Association.
Dr. Thomas Joyce	Representing the Pacific Coast Surgical Association.

The following officers were elected:

Chairman—Dr. Evarts A. Graham.
Vice-Chairman—Dr. Allen O. Whipple.
Secretary-Treasurer—Dr. J. Stewart Rodman.

Two groups of candidates are recognized for qualification by the Board.

(A) Those who have already amply demonstrated their fitness as trained specialists in surgery.

(B) Those who, having met the general and special requirements exacted by the Board, successfully pass its qualifying examination.

The first of these groups, the Founders Group, upon invitation by the Board will be chosen from the following:

(1) Professors and Associate Professors of Surgery in approved medical schools in the United States and Canada.

(2) Those who for 15 years prior to the Board's organization have limited their practice to surgery.

Submitted for publication May 7, 1937.

(3) Members of the American Surgical Association, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association and the New England Surgical Society, who were in good standing January 9, 1937.

All applications for the Founders Group must be received within two years of the Board's organization, January 9, 1937. No candidates for the Founders Group will be considered after that date.

Requirements for those to be qualified by examination will be as follows:

(1) Graduation from a medical school of the United States or Canada recognized by the Council on Medical Education and Hospitals of the A. M. A. or graduation from an approved foreign school.

(2) Completion of an internship of not less than one year in a hospital approved by the same Council, or its equivalent in the opinion of the Board.

(3) *Special Training.* A further period of graduate work of not less than three years devoted to surgery taken in a recognized graduate school of medicine or in a hospital or under the sponsorship accredited by the American Board of Surgery for the training of surgeons. This peroid of special training shall be of such character that the relation of the basic sciences of anatomy, physiology, pathology, bacteriology and biochemistry is emphasized. Knowledge of these sciences as applied to clinical surgery will be required in the examination. Adequate operative experience in which the candidate has assumed the whole responsibility will be required. An additional period of not less than two years of study or practice in surgery.

(4) The candidate must present to the Board sufficient evidence of good moral character as to justify it in the belief that he will not engage in fee splitting and other dishonest practices.

It is expected that the Board, with the assistance and cooperation of the American Medical Association and the American College of Surgeons, will be able to increase the facilities which now exist for the adequate training of young surgeons by means of residencies, fellowships, *etc.*, in suitable hospitals.

The above requirements, especially those referring to surgical training, are subject to change from time to time as the existing opportunities for training in this field of specialization may be broadened.

The qualifying examination will be divided into two parts: Part I, written, and Part II, clinical, bedside and practical. The written part, Part I, will concern itself with general surgical problems and with the clinical application of the basic sciences of surgery to these problems. This examination will cover a period of three hours each and will be held simultaneously in as many centers as are necessary to accommodate the number of applicants who are eligible. Part II is entirely oral and will also concern itself, in the main, with general surgery and, as stated for Part I, clinical application of the basic sciences to the clinical problem represented. In addition to this, in Part II, an examination will be given to test the candidate's knowledge of operative surgery, roentgen ray film interpretation and the principles and application of surgical anesthesia. This examination will be held in as many centers as the Board may determine necessary to accommodate the eligible candidates. Reexaminations will be allowed providing one year shall elapse between examinations.

The fee for Group A, the Founders Group, shall be $25. The fee for Group B shall be $75, payable as follows: $5 for registration fee, which shall be returned if the candidate is not accepted for examination; $20 for Part I; and $50 for Part II. The same fee will be required for each reexamination. Once the candidate has become qualified, he will have no further financial obligation to the Board.

This Board is a non-profit organization. All fees will be used, after a reasonable

amount is set aside for necessary expenses in maintaining its office, conducting examinations, *etc.*, to aid in improving existing opportunities for the training of the surgeon.

A certificate attesting to a candidate's qualifications in surgery after meeting the requirements of the Board will be issued, having been signed by its officers.

Any certificate issued by the Board shall be subject to revocation by the Board at any time in case it shall determine in its sole judgment, that a candidate, who ha received a certificate, either was not properly qualified to receive it or has become disqualified since its receipt.

The Board will hold its first examination (Part I, written) on September 20, 1937. All inquiries concerning applications for this examination should be received by the secretary's office promptly.

Requests for booklets of information, application blanks, and other information should be addressed to the Secretary—Dr. J. Stewart Rodman, 225 South 15th St., Philadelphia, Pennsylvania.

J. Stewart Rodman, M.D.
Secretary.

EPHRAIM McDOWELL MEMORIAL

For some years you have been good enough to continue me as a committee of one to cooperate with the Kentucky State Medical Association in securing the Ephraim McDowell home for conversion into a permanent shrine. Until now I have been unable to report progress, but am happy to state that the project is in process of completion. In May of 1935, the Kentucky State Medical Association erected a memorial to Jane Todd Crawford, the patient upon whom Doctor McDowell performed his epoch-making operation. At this meeting the then President of the Kentucky State Medical Association appointed a Committee consisting of Drs. A. T. McCormack, Marshall McDowell, Louis Frank, Charles Vance and Irvin Abell to negotiate for the purchase of the property and to raise funds for its payment. I am happy to report that the property was purchased by the Kentucky State Medical Association, November 22, 1935, and in turn deeded to the Kentucky State Department of Public Property under supervision of the State Parks Board. This latter was done in order to secure an appropriation from the P. W. A. of $13,500 for restoration purposes. To the purchase price of $15,000 the then owners of the property contributed $5,000 as a memorial to their father, the late Doctor Wiesiger, leaving $10,000 to be raised in donations from the profession of the country. I am glad to state that this amount has been fully subscribed and particularly pleased to note that 51 members of the Southern Surgical Association have contributed $3,230 to the fund; this does not include the donations of the Kentucky members of the Southern Surgical Association, since these naturally have been listed with those from the members of the Kentucky State Medical Association. Our books are still open in the hope that we will secure additional funds for the purpose of purchasing an adjoining piece of property which investigation shows to have been Doctor McDowell's Apothecary Shop, and further for the erection of a statue of Doctor McDowell in the garden of the home.

The work of restoration began August 10, 1936, and we are hoping to have it completed during the coming year. By deeding the property to the Kentucky State Department of Public Property, the perpetuity of the Memorial is assured. As this has become known, we have been able to secure mementoes and relics of Doctor McDowell for preservation in the home.

IRVIN ABELL

MEMOIRS

WILLIAM OLIVER FLOYD

1880–1936

AT THE 1935 meeting of the Southern Surgical Association at Hot Springs, Doctor Floyd was taken ill with influenza, which culminated in pneumonia, from which he died January 12, 1936, at the age of 56. He was well known as a surgeon throughout his native state of Tennessee and

WILLIAM OLIVER FLOYD, M.D.

contiguous territory; a man of ripe judgment and sincere devotion to duty, and particularly solicitous about the welfare of his patients.

He received his Bachelor of Science degree from Peabody College in

1907, and was graduated in medicine from the University of Nashville in 1910. He interned at St. Thomas Hospital and after that had been identified with it continuously as Assistant Surgeon and later Visiting Surgeon, the day of his death marking the completion of his year's service as President of the Staff. He was a Fellow of the American College of Surgeons and had been a member of the surgical staff of Vanderbilt Hospital and the Nashville General Hospital. He was one of the assistants to the Chair of Surgery at Vanderbilt University.

Doctor Floyd was a man of unusual common sense, the basis certainly of all scientific and practical surgery. With increasing experience he developed a ripe surgical judgment; any error was always on the side of conservativeness. He never did anything questionable or risky for a patient if he could avoid it. From a large volume he remembered the essentials and profited by every item of experience. He was slow to take on the new, but he held to the well-tried. He was really at his best during the emergencies that arose during the course of an operation, always cool, deliberate, but extremely resourceful. In diagnosis he had an uncanny faculty for picking out the important and sorting it out from the nonessential. He never lost sight of the most probable explanation of the condition. Being a man of rare discernment he was able to winnow the functional from the organic and was never allured by the neurasthenic.

His interest in roentgenology was early and at all times sustained. He had the unusual opportunity of comparing all of his roentgenologic studies of the viscera with the pathologic condition found at operation, which is a rare combination and which he utilized to the full. This gave his interpretation of roentgenograms exceptional value.

While very unobtrusive, Doctor Floyd was sincere and dignified, so that those who knew him came to love him and admire him as a man of character, inherent honesty, and rare conscientiousness. He was patience personified. Disraeli said, "Patience is a necessary ingredient of genius." These attributes, together with his tolerance and imperturbability, made him a splendid type of surgeon. The esteem in which he was held was most unusual and an example of what honesty, steadfastness of purpose, and high ideals in surgery will accomplish.

He made a number of contributions upon diseases of the gallbladder and ducts. In a large experience he encountered many bizarre and unusual cases which were reported. He had written upon the surgical conditions of the colon, goiter, and made a comprehensive study of the end-results in our cases of breast cancer.

Doctor Floyd never sought a confidence, but if it were given to him he held it inviolate.

In a memorial tribute his colleagues said of him, "In his blood ran the essence of humanitarianism blended with skill and understanding. To the many his ministrations dispelled despair and became a beacon of hope and renewed energy. As a man he lived simply but richly; he thought clearly and without prejudice; he was modest and unostentatious. He served justice and spoke no evil."

WILLIAM D. HAGGARD

CHARLES JEFFERSON MILLER

1874–1936

"His wise, rare smile is sweet with certainties,
And seems in all his patients to compel
Such love and faith as failure cannot quell;
We hold him for another Herakles,
Battling with custom, prejudice, disease,
As once the son of Zeus with Death and Hell."—*William Ernest Henley.*

It is difficult for one closely associated with Dr. C. Jeff Miller for many years to become his biographer, because it is hard to strike the perfect mean

Charles Jefferson Miller, M.D.

between a chronicle of the facts of his life and achievements and the deeper chords of personal reverence for a departed Chief and friend. Not only for this reason is it difficult, but because "Doctor Jeff" spoke so seldom of himself and allowed so little praise of himself, that, almost, his voice is

heard in deprecation of the homage we wish to pay his memory. To those of his associates who knew him well, to his patients, and to many of his old pupils, it would doubtless be equally hard. They held the man and the surgeon in deep veneration; he inspired them with confidence in his knowledge, faith in his wisdom, and dependence upon his unbiased judgment. Daily, letters came to Dr. C. Jeff Miller—from doctors, asking for advice on problems of practice, from patients who seemed to think he could diagnose and alleviate their most minute difficulties—sight unseen, and from former pupils seeking his advice on medical questions.

Dr. Charles Jefferson Miller was born on February 9, 1874, at Winchester, Tenn., and passed his early life on his father's farm. Tennessee might well claim credit for his education, for all his undergraduate years were spent there: in the primary rural schools near his home; Terrill College, Sewanee, which he attended on a scholarship and from which he was graduated; and then the University of Tennessee, where he earned his M.D. degree in 1893.

At no time in his career was he associated in practice with an older man; unaided except by the admiration, friendship, and respect of his confreres, he established a name for himself. Although he was very young when he first came to New Orleans a few years after his graduation, his reputation as a surgeon of brilliance and sound judgment grew rapidly. His fame spread not only in New Orleans and in Louisiana, but throughout the South, and he became eventually one of the leaders in his specialty with a professional standing not only national, but international.

Almost from the beginning, his interest centered in gynecology. As early as 1898, he was Visiting Gynecologist to Charity Hospital, New Orleans, and Chief of Clinic to the Chair of Gynecology and Obstetrics of Tulane University, under Dr. Ernest S. Lewis, as well as Lecturer on Clinical and Minor Gynecology at the New Orleans Polyclinic. In 1910, he was appointed Professor of Clinical Obstetrics and Clinical Gynecology in the Tulane University of Louisiana, and in 1925, Professor of Gynecology and Obstetrics, becoming head of the Department in 1927, which position he held at the time of his death on March 21, 1936.

Dr. C. Jeff Miller exerted perhaps the most universal influence upon gynecology in the South of any man of his time. Through his teachings in the classroom and clinic, his writings, and his personal influence, he became the apostle of modern trends in gynecology at a period when many discordant voices were preaching confusing theories.

But he was not a prophet without honor in his own country. In 1897, a few years after his advent to New Orleans, he was elected Secretary of the Orleans Parish Medical Society, and in 1906, its President. He was President of the Howard Memorial Library, New Orleans; a member of the Board of Control of the Louisiana Leper Home until it was taken over by the Federal Government. Besides being Chief of a gynecologic service at Charity Hospital, he was also Chief of that department at Touro Infirm-

ary, Chairman of the executive committee of the staff of Touro Infirmary, and a member of the Medical Advisory Board of Flint-Goodridge Hospital of Dillard University, a hospital for Negroes. He accepted no office merely for the civic, social, or business prestige it might carry; his interest centered in his profession and in those activities which were directly associated with it.

The esteem with which he was regarded all over the country is concretely evidenced by the honors he received. He was President of the American Gynecologic Society in 1928–1929; President of the American Gynecologic Club in 1927; Secretary of the Section on Obstetrics, Gynecology and Abdominal Surgery of the American Medical Association in 1908, and Chairman in 1912; President of the American College of Surgeons in 1930, and a member of the Board of Regents from 1926 until his death; President of the Southeastern Surgical Congress in 1936.

In 1898, Doctor Miller became a Fellow of the Southern Surgical Association; in 1920, Vice-President, and, in 1922, President. The work of this organization was always one of his chief interests. He attended the annual meetings faithfully; press of work sometimes kept him away, but it was only some unusual circumstance that prevented his going, for he looked forward to the opportunity these gatherings afforded of renewing old friendships as much as he did to the opportunity of witnessing the scientific attainments of the members of the Association, in which he took an active part.

Doctor Miller was the author of two widely used text-books: An Introduction to Gynecology and Clinical Gynecology, as well as of innumerable articles on scientific subjects and many of a medico-literary nature. He kept up with everything written on his specialty in the leading journals of America and abroad. His knowledge of the history of medicine was minute, and, always an interesting conversationalist, he was especially interesting on this subject. He was also an avid general reader, and many of his hours free from the rounds of professional routine and teaching were spent with books.

He enjoyed lecturing to his students and could never be persuaded to give up this arduous part of his life's work. He was especially interested in perfecting the standards of training both for undergraduates and for postgraduates who intended specializing in surgery. But he urged always in the classroom and in his writings that "the humanities of medicine" be not forgotten, or as he said in his inaugural address as President of the Southern Surgical Association: "Let us always be found defending the proposition that medicine is more than a science, that a skillful physician must be more than a scientist, that he must possess other knowledge that we usually refer to as the art of medicine or surgery, and that the possession of one without the other may be equally harmful."

And it was in that same address, 15 years ago, that he sounded an almost prophetic note of warning to the profession in regard to socialized medicine.

In 1930, Sewanee, recognizing his work toward the advancement of the science and art of medicine and the glory he had brought to his Alma Mater,

conferred upon him the degree of Doctor of Science "for his contributions to medical science."

We who knew him well lament the passing of a friend, an influence in our daily lives, a dearly loved preceptor. The medical world mourns the loss of one of its foremost surgeons and gynecologists, a great teacher, a captain in the march of medical progress. But we who suffered a personal loss in his going hold undying memories of him in our hearts. And in the medical world his teachings still live; his achievements are not forgotten. The words of Tennyson read at his last rites sound his last instructions to us:

"Sunset and evening star,
And one clear call for me!
And may there be no moaning of the bar
When I put out to sea."

CURTIS H. TYRONE

SOUTHGATE LEIGH

1864-1936

DOCTOR SOUTHGATE LEIGH was born May 21, 1864, in Campbell County, Va., and died in Norfolk, Va., March 6, 1936. An only son of John Perviance and Fanny Cowdrey Leigh, he was left an orphan early in life and

SOUTHGATE LEIGH, M.D.

made his home with an aunt, Mrs. Pegram, for whom, in loving memory, he named the admirable hospital he built and operated to the time of his death. His early education was in the private school of Mr. Galt. After an academic and medical course at the University of Virginia under such able teachers as Doctors Cabell, Mallet, Towles, and Dabney, he received the degree in medicine, in 1888. Going to New York, he matriculated at the College of Physicians and Surgeons, Columbia, and was given a second degree of M.D. His internship was at Mt. Sinai Hospital where he was

directly under that eminent surgeon Dr. Arpad Gerster, from whom he received honored consideration and was given special and prolonged service.

A year in Vienna rounded out a well grounded medical education, and he returned to Norfolk, in 1893, and entered the practice of his profession.

Endowed with abundant energy, he sought an outlet in clinical work and was largely instrumental in organizing the first Clinic at St. Vincent's Hospital. He helped energize the old Retreat for the Sick, afterward the Norfolk Protestant Hospital, and was, by his vision, largely responsible for its present excellent topographic location. Later he built and administered the Sarah Leigh hospital and Clinic which now is in the process of being made a memorial to his memory through the activities of his many admiring friends. His driving energy led him to interest himself in the civic affairs of the city in which he held various important positions and in recognition of which he received the medal as "First Citizen" from the Cosmopolitan Club.

He was a faithful attendant at the meetings of many medical societies and was honored by the presidencies of the Norfolk County Medical Society, The Seaboard Medical Association, The Southern States Association of Railway Surgeons, Norfolk and Western Association of Railway Surgeons, Chesapeake and Ohio Association of Railway Surgeons and the Medical Society of Virginia.

He gave generously of his time and talents to his many patients, regardless of pecuniary reward, and also to his civic activities. His death came suddenly from cerebral hemorrhage while making a report of his successful effort to increase the membership of the Association of Commerce. As an evidence of the esteem in which he was held the funeral was attended by a very large gathering from all walks of the citizenry of the City.

Married, in 1905, to Alice Creekmore, she survives with two sons—Dr. Southgate Leigh, Jr., and Watkins Leigh—and two daughters: Mrs. Martin H. Capps and Mrs. Lester M. Minkel, both of New Jersey.

LOMAX GWATHMEY

EDITORIAL ADDRESS

Original typed manuscripts and illustrations submitted to this Journal should be forwarded prepaid, at the author's risk, to the Chairman of the Editorial Board of the ANNALS OF SURGERY

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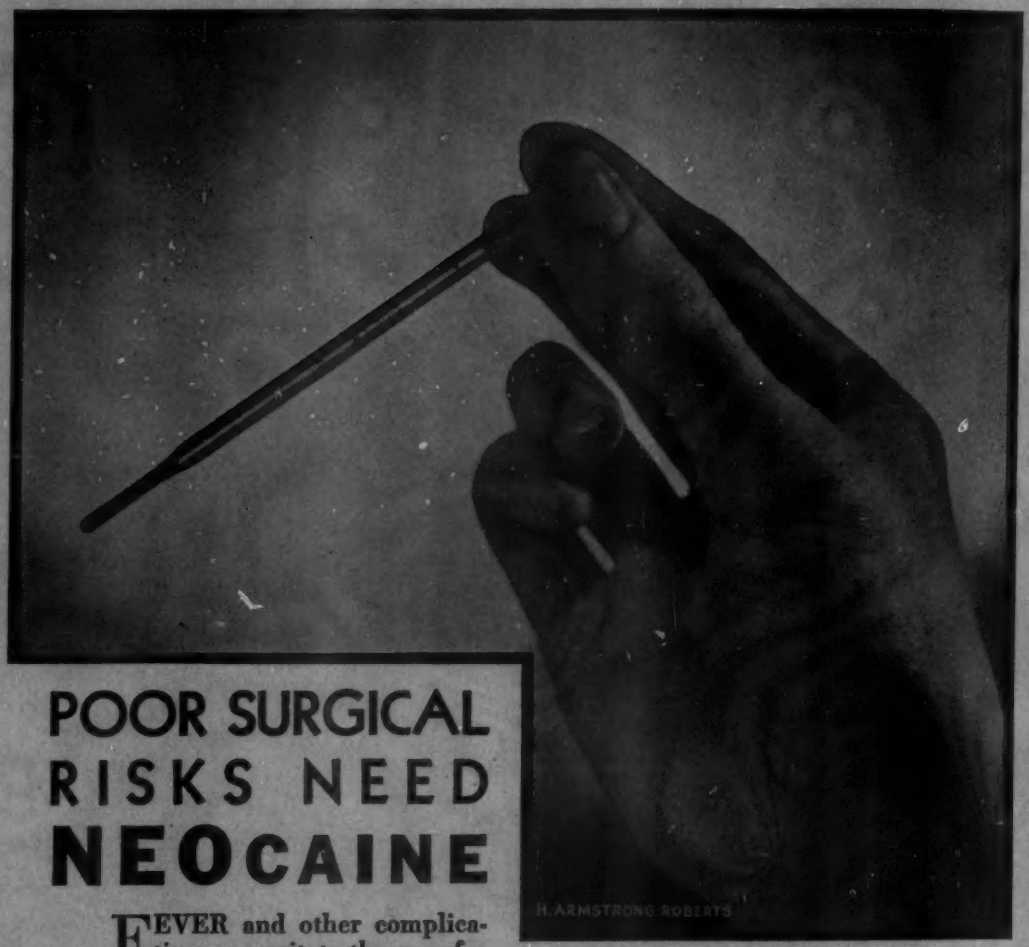

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